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TEXT-BOOK
ON
NERVOUS DISEASES

BURR

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ON
NERVOUS DISEASES

BY

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GENERAL DIAGNOSTICS
OF
NERVOUS DISEASES

VOL. II.

V

DISEASES OF THE BRAIN

(*Continued.*)

2. DISEASES OF THE MEMBRANES OF THE BRAIN AND THE SPINAL CORD

BY

HUGO STARCK (Karlsruhe)

As the diseases of the membranes of the spinal cord with the exception of *hypertrophic cervical pachymeningitis* and *syphilitic meningitis*, which are discussed elsewhere, can not lay claim to any special clinical significance, they will not be considered separately here. They will be considered here only in so far as they depend upon diseases of the cerebral meninges, or when they form a partial symptom of such disease.

Tumors and hæmatomata of the dura mater are also discussed elsewhere.

Diseases of the Pia-arachnoid of the Brain and the Spinal Cord (*Leptomeningitis*)

We distinguish an *acute and a chronic leptomeningitis.*

Acute leptomeningitis manifests itself in various forms, as 1. *Primary purulent leptomeningitis* (*epidemic cerebro-spinal meningitis*); 2. *The secondary purulent forms:* (*a*) *consecutive*, (*b*) *traumatic*, (*c*) *metastatic* resp. septic leptomeningitis; 3. *tubercular meningitis* and 4. *serous meningitis.* It is probable, however, that this latter form may also appear as an independent disease, and thus bear a primary character (see later).

Chronic leptomeningitis is only in rare cases to be regarded as a primary disease; it usually develops upon a syphilitic foundation.

Syphilitic leptomeningitis is treated in the chapter on syphilitic diseases of the central nervous system.

1. ACUTE LEPTOMENINGITIS

A. Primary Purulent Leptomeningitis

Epidemic meningitis alone seems to be a disease sui generis—one which is caused by a specific micro-organism, for the remaining forms are *not uniform ætiologically*, i. e., each one is not caused by a specific micro-organism. They may be caused by various micro-organisms and only the ways or means by which these reach the membranes are different.

Clinical observation also shows that the epidemic form occupies a special position among the varieties mentioned, in that, leaving its epidemic dissemination out of consideration, it shows a more sharply defined clinical picture than the others.

EPIDEMIC CEREBRO-SPINAL MENINGITIS

Epidemiology.—Epidemic cerebro-spinal meningitis, also called *epidemic nuchal rigidity* (Genickstarre), has been known only since the beginning of the last century. In the spring of 1805 this hitherto-unknown disease first appeared in *Geneva*, and since that date the infection has slowly spread more and more widely, until gradually no civilized nation has remained unspared. In the second half of the last century, it manifested itself in *Germany*, and at present appears in all districts occasionally. (It appeared in Medford, Mass., at the same time as at Geneva, and was described by Danielson and Mann. Many epidemics have occurred in America since.)

As a rule, the epidemics are not very extensive, and even in areas with a large population, the disease claims only a victim here and there (in large cities only 20–50). This peculiarity of the disease may safely be regarded as a characteristic feature of the epidemic. It usually makes its appearance in winter and spring. The disease seems to have a predilection for appearing among *bodies of troops* (*barracks*) and there seems to affect especially recruits. Frequently, however, *children*, particularly *infants*, are mainly affected. According to the statistics of *Hirsch* (Danzig), out of 779 patients, 8% were children under 10 years, and 25% were infants. The disease rarely affects persons over 40 years of age.

Concerning the *conditions of its dissemination* but little is known. Direct infection seems scarcely ever to occur; possibly domestic animals, as they have frequently been known to be affected with purulent meningitis, may play a mediatory rôle.

Sporadic cases of the same nature as those found in epidemics, have also been observed; but it must be remembered that in epidemics the cases are usually widely scattered, that their epidemic character may ultimately be obliterated (see also below).

The *generator of the disease* is the *meningococcus intracellularis*, which was found in 1887 by *Weichselbaum* in cadavers, and by *Heubner* in the fluid withdrawn by lumbar puncture from the living.

It must be emphasized here, that this view of the **specific ætiology** of epidemic cerebro-spinal meningitis has not been universally accepted. *Fraenkel*, *Stadelmann* and others, have found in purulent meningitis, which clinically in no wise differed from the epidemic form, *Fraenkel's pneumococcus*. A number of authors are therefore of the opinion that epidemic meningitis may be produced by several, or at any rate by these two micro-organisms. It is a fact that in the epidemic form a mixed infection is also occasionally observed, in which the pneumococcus plays a part. *Heubner*, however, believes that the epidemic cerebro-spinal meningitis is produced only by the meningococcus, and that the pneumococcus is to be regarded as the pathogenetic agency in sporadic purulent meningitis.

The *meningococcus* is chiefly found in the pus cells, but also free in the fluid portions of the exudate. In the cells it resembles the gonococcus and is arranged in pairs, which lie with their broad sides against each other (in contradistinction to the pneumococci). Frequently four of them lie together. *Heubner* was able to reproduce purulent meningitis in goats with the meningococcus.

The cocci are often exceedingly scanty and difficult to find in the preparations; usually they are *Gram-negative.* On agar they grow in luxuriant colonies.

Frequently the meningococcus is not to be obtained pure in epidemic cerebro-spinal meningitis, but in mixed cultures particularly with *Fraenkel's pneumococcus.* In addition, staphylococci, streptococci, and the bacilli of influenza have been found associated with it.

Our knowledge concerning the *mode of infection* is still very vague. *Weigert and Struempell* have found the meningococcus in autopsies, in the naso-pharyngeal mucus, and in the accessory nasal cavities; but even in healthy persons it was found in these places. It is therefore assumed, that from there it reaches the subarachnoid space by way of the lymphatics. *Westenhoeffer* regards the pharyngeal tonsil as the source of the infection, and believes that it enters through the naso-pharynx. Many circumstances speak for a hematogenous infection of the meninges.

In many cases no cause can be assigned for the sudden exuberant growth of the meningococci; but in other cases, exposure to cold, getting drenched, trauma, and severe mental over-exertion seem to have acted the rôle of a determining cause (*Gelegenheitsursache*).

Pathological Anatomy.—When the skull cap is removed, the dura appears tense, its internal surface dry, the sinus fairly bulging with blood. The convolutions are flattened and broadened, the fissures contracted; signs of *intracranial pressure.*

The condition of the *leptomeninx* differs according to the stages of the disease. In cases taking a violent course, which lead in a very short time to death, besides a high degree of venous hyperæmia, only a turbid swelling is observed. As yet, there is no indication of pus formation, except that the dark blue veins are accompanied by narrow light yellow to greenish stripes consisting of a sero-purulent exudate. Sometimes large, blotch-like accumulations of such exudations are found in circumscribed spots.

In other cases, particularly in those in which the disease has already existed several days, the accumulation of pus may assume remarkable dimensions, so that the whole surface may be covered by a layer of pus.

Much oftener, however, more localized accumulations of pus take place, so that the cerebellum and the fore-brain, f. e., are covered with pus, while other parts are almost entirely free from it; or, in still other cases, the cranial nerves are embedded in a mass of pus, while the hemispheres remain free. It is seldom that the process is restricted to the membranes alone; usually the pus permeates the *substance of the brain and spinal cord* to a greater or less extent.

In the *spinal membranes*, the exudate chiefly collects in the lumbar segment, and most frequently on the posterior surface.

The *ventricles* contain a cloudy purulent exudate; in the later stages *hydrocephalus* develops.

When the disease does not run an acute course to death, it will be found that after a few weeks the meninges are thickened, as well as shrunken, the ventricles widely dilated; at the base a thick rind-like layer of pus is covered by a more or less clear liquid. The ependyma shows warty thickenings.

Neighboring organs, the labyrinth and the eye, which by means of the lymphatics communicate with the subarachnoid space, often fall victims to the purulent infection.

Clinical Picture.—During the course of an epidemic of cerebro-spinal meningitis, a two year old child falls ill with mild general symptoms, such as headache, dizziness, transient nausea; there is likewise a slight pharyngitis. On the third day, a sudden exacerbation and a chill occur, followed by a temperature of 103, violent headache, great dizziness, vomiting, prostration. On the next day, herpes are seen upon the lips; the head is buried deeply in the pillow; every attempt to bend it forward, produces the most violent pains at the nape of the neck; *the neck is rigid.* The headache has now become unbearable, so that the child moans and screams aloud. All food is refused; there is considerable irritability; day-light is painful to the eyes and increases the headache. Great motor restlessness renders all sleep impossible. The fever is somewhat decreased, the temperature ceases to rise beyond 102 degrees, and is remittent. At times it seems as if amelioration would set in, but one soon becomes convinced of the contrary; the face is flushed; the tongue and lips are dry; the general super-sensitiveness is still more increased. The pulse is frequent, feeble, and irregular; respiration is accelerated. The entire spinal column has become rigid and exceedingly susceptible to pressure; the masseters are spasmodically contracted (*trismus*). By the end of the first week, the clinical picture is changed, in that the child has now become apathetic; it does not move any more, and only now and then touches its neck and screams in its sopor; while up to this time, the sensorium was unaffected, stupefaction now supervenes.

Death comes in deep coma on the tenth day.

2. A young man of twenty-three, without any premonitory indications, is seized in vigorous health by chills, violent headache, vertigo, and repeated

vomiting. The temperature rises to above 103 degrees. The evening examination reveals fixed rigidity of the spinal column (opisthotonos and stiffness of the neck), great hyper-sensitiveness of the whole body, and an enlargement of the spleen with tenderness on pressure. On the next day, herpes appears upon the lips and the body is covered with an urticaria-like exanthem. The violent incipient symptoms have somewhat subsided, but the sensorium has become benumbed. At the end of the first week a marked change in the symptoms occurs; during the day consciousness varies, clear in the morning, stuporous toward evening; the general condition is improved; the headache appears sporadically, but when present is always violent; a relatively good condition is followed by sudden vomiting cerebral in type; the rigidity of the neck continues unchanged; all food is absolutely refused. The temperature fluctuates between 100 and 102.

This condition lasts through nearly the entire second week, although the sensorium has by now become almost wholly clear. At the end of the second week, optic neuritis becomes apparent, whereas up to this time the background of the eye had remained normal, and during the third week an iritis with hypopyon sets in, which in the next ten days forms a serious complication of the primary affection. Just as the eye affection seems to be receding the patient complains of increased dizziness and pain in the ears. An affection of the labyrinth is developing, which is characterized at first by an unilateral, but later bilateral, tinnitus aurium and most severe vertigo. The fever has disappeared almost entirely; the general condition, aside from the persisting anorexia, is quite tolerable; in the sixth week the patient may get out of bed and move about when renewed general pressure in the head appears. While up to this time the disease picture was not complicated by any spasms, the latter now set in from time to time in the extremities; the psychic functions decrease; as a result of the great weakness in his legs the patient is again confined to bed. The supposition that a chronic *hydrocephalus* has developed, now becomes a certainty; marked emaciation, paralysis of the sphincters, serious bed-sores lead to death eight weeks after the onset of the disease.

Symptomatology.—Even though the individual clinical pictures show quite considerable differences, yet the disease shows so many common features, prominent characteristic symptoms, that the diagnosis as a rule is not difficult. As the chief characteristic feature we note, that during nearly the whole course of the *acute stage*, *symptoms of irritation* occupy the foreground, while *symptoms of paralysis* are either entirely lacking until death terminates the scene, or manifest themselves only in the last stage.

The onset is usually sudden; vigorous health is abruptly interrupted by a violent chill with high fever; synchronously most violent headaches and vertigo, vomiting, intolerance to light, set in. In other, rarer, cases, the disease proper is preceded by *prodromes:* a cold; pharyngitis; slight angina;

conjunctivitis, associated with a feeling of general discomfort; chilliness; slight dizziness; restlessness; and headache. After two or three days the real *outbreak of the disease* is marked by sudden violent symptoms, such as headache, vertigo, rigidity of the neck; the change, however, may also be gradual.

In children the disease is not infrequently introduced by general *convulsions.*

The *cardinal symptoms* of the developed disease are, then, *headache, vertigo, vomiting, nuchal rigidity, and a clouding of consciousness.*

One of the early symptoms, which is frequently the first to arouse a suspicion of meningitis, is the *headache* which sets in sometimes suddenly, sometimes insidiously, but which even in the first few days (occasionally in the first few hours) becomes so intense as to be intolerable. It must be regarded, however, as characteristic, that although the headache continues incessantly, it has *paroxysms of exacerbation* and remissions in rapid succession. It is most frequently localized in the occiput or in the forehead or temples though it may involve the entire head. Even in the later stages and during convalescence, attacks of pain may again manifest themselves occasionally.

Vertigo is scarcely ever absent, and particularly in the first few days is never lacking. When it sets in during convalescence, it ought invariably to arouse a suspicion of a *late complication in the auditory apparatus.*

The *vomiting* is cerebral in character. Very early, often only a few hours after the onset of the disease, the *neck* becomes rigid (therefore the German name of the disease: *Genickstarre*) and painful; the head, as a result of the tonic contracture of the muscles of the neck, is arched backward, and fairly bores into the pillow; every attempt to bring the chin nearer the breast is accompanied by violent pains in the neck; *in no other form of meningitis does the rigidity of the neck stand so prominently in the foreground of the affection as in epidemic cerebro-spinal meningitis.* Generally the rigidity is not confined to the neck; the whole spinal column is rigid and tender to pressure, and as a result of contracture of the long extensors of the back is curved as in lordosis (*opisthotonos*).

Consciousness is almost always clouded, but is subject to great and unexpected fluctuations. Only in light cases it remains free until recovery; in serious cases it lapses into somnolence, sopor, and coma. As a rule a slight stupor exists, which may, however, be interrupted by violent delirium.

Convulsions occur more frequently in children than in adults. They may assume an *epileptic type*, are often confined to one extremity or one half of the body, and are followed in rare cases by *paralysis* of the part concerned. Occasionally, clonic twitchings in the areas of the facial and hypoglossus nerves and of the muscles of the eye are seen.

A sign of *hypertonia of the musculature* is grinding of the teeth, *trismus,* and the spasmodic boat-like retraction of the abdomen; it occasionally seizes the musculature of the entire body, so that the patient appears to be affected with tetanus.

Accordingly, a general *exaggeration of the reflexes* is noted in most cases. The cutaneous reflexes are almost invariably increased; but this does not apply to the tendon reflexes which may vary greatly.

The *Babinski* reflex is occasionally present together with a considerable increase of the patellar reflex.

Kernig's sign (impossibility of a right-angled flexion, inability to flex the extended leg while lying on the back so as to form a right angle, or to extend the leg when sitting, on account of contracture of the flexors) is also an almost invariable symptom (cf. Fig. 161, p. 580). In the last stage the reflexes disappear altogether. (Kernig's sign is sometimes present in pneumonia in children with meningitic symptoms, and even in typhoid fever.)

As already mentioned, *symptoms of paralysis* as compared with those of irritation, remain quite in the background. At any rate, in the first stages of the disease they are exceptional. In a few instances, however, permanent monoplegia, hemiplegia or paraplegia have been observed.

General *sensibility* deviates from the norm in so far as an *extreme sensitiveness* to all external stimulation exists; pains are felt everywhere: in the arms, the legs, and the joints; the susceptibility is especially increased in a few circumscribed spots, as the heels, toes, and where the osseous portion of the ribs passes over into the cartilaginous part. The pains cause the unfortunate patients to continually wail and moan, and to make the most unnatural writhings and painful distortions of the face.

Not only touch perception is increased to the utmost; *light and noise are felt as pain*, and the *nerves of taste and smell* are likewise *super-sensitive.*

Pronounced *anæsthesias* are rarely found; on the other hand the patient complains of disturbing *paræsthesias* in addition to the manifestations of pain mentioned.

The *vaso-motor nerves* are also abnormally excitable; the slightest cutaneous irritations often produce long continued erythema and urticaria-like wheals. The pressure of the bed-clothes suffices to induce an intense erythema.

In the majority of cases a *labial herpes* develops sometime between the second and the fifth day. In the course of the disease the most varied *exanthemata* are observed: stipple-like spots, like the rash of typhoid fever, urticarious, measle- and scarlet fever-like exanthemata. Erythema exsudativum multiforme and hemorrhagic infiltrations may also develop in the later stages.

Generally speaking, the frequency of the pulse corresponds to the temperature. Frequently it is found to be *lessened;* and irregularities form part of the typical picture. The *temperature* does not exhibit a typical course.

After the first acute rise it usually abates somewhat, and then takes an irregular, often a remitting course. In subacute cases, periods of slight fever may alternate with others entirely free from fever.

One of the most unpleasant, but most frequent and persistent symptoms relative to the *digestive organs* is *anorexia*, an absolute aversion to taking any food whatsoever, which leads to extreme emaciation and which is often responsible for the unfavorable outcome. The tongue may be dry, fuliginous for weeks; also the lips; the naso-pharynx is very dry as well. In addition to vomiting (which mostly bears a cerebral character), the patients are affected with troublesome retching or hiccough.

The *bronchial organs*, aside from the prodromal symptoms, are seldom involved in the morbid process. (If pneumonia occurs, death practically always follows.)

(Pericarditis with few adhesions and slight effusion may be present. The absence of physical signs makes diagnosis difficult.)

The kidneys also usually remain normal.

On the other hand, in the course of the disease *joint affections*, inflammatory swellings, frequently manifest themselves, which, if they appear at the beginning of the disease, at first make one think of an acute polyarthritis.

Still another important group of symptoms should be mentioned, namely, disorders of the *cerebral nerves*. It is true that they may be entirely absent in acute cases; they are found rather in cases taking a subacute and chronic course, and most frequently in those forms of meningitis caused by the pneumococcus. But since at times they may also be found in cases of pure meningococcic meningitis, they are to be described here as *complications*. I wish to state beforehand that the frequency and nature of the complications differ in the various epidemics. Since the subarachnoid space communicates through the lymphatics with the various accessory cavities, it is only natural that these should sometimes be affected through the continuity of the inflammation, but in other cases it is more a matter of metastatic processes. Probably the eye most frequently participates in the complication. At first the pupils are narrow, later dilated or unequal in size; nystagmus is frequently observed; and paralysis of the muscles of the eye also occurs, although more rarely than in the other forms of meningitis, especially the tubercular (cf. Fig. 160, p. 579). *Optic neuritis* and retinitis are frequent complications; more dangerous complications, such as keratitis, iridocyclitis, hypopyon, and panophthalmitis (*Axenfeld*) also occur, and eventually lead to total blindness.

Complications affecting the *ear*, such as otitis media, with perforations of the tympanum, and diseases of the labyrinth, which may be followed by deafness or in children by deaf-mutism, are not infrequent.

Finally, the *sequelæ* should be noted. They not infrequently assume a permanent character. Above all, serious *nervous disorders* should be mentioned—disorders which may continue throughout the remainder of life such as tinnitus aurium, vertigo, headache, weak memory, fatigue of mental concentration, etc. Permanent *paralyses of the cerebral nerves and of those of the extremities* belong also to this class. We have already mentioned above deafness and blindness.

Another of the most dreaded sequelæ is *chronic hydrocephalus*, which may supervene even during convalescence. In small children the size of the head may grow larger under the eyes of the observer and assume immense dimensions. In adults the nervous symptoms before mentioned lead to the diagnosis. *Eichhorst* has observed an almost sudden death in several of these cases. Children gradually become *imbecilic;* very rarely the symptoms disappear with the years. (Compare the chapter on hydrocephalus.)

Course.—Several types may be distinguished according to the *course of the disease.* Fulminant *cerebro-spinal meningitis* (*acutissima or siderans*) is marked by death resulting even in a few hours. Autopsy reveals in such cases only hyperæmia and opacity in the membranes, but no pus as yet.

Cases which, like apoplexy, begin with unconsciousness and paralysis have been designated *meningitis cerebrospinalis apoplectiformis.*

In contradistinction to these more violent forms stand the *rudimentary, abortive cases*, which only in the course of an epidemic are interpreted correctly. The disease manifests itself only by headache, dizziness, lassitude, supersensibility. A slight degree of rigidity of the neck may sometimes obtain in these cases. Recovery follows in from one to two weeks.

Lastly, the *intermittent* form should be mentioned, in which all symptoms rise and fall after the manner of a malarial affection, while in the time free from attacks, the patient feels well and has no fever. Formerly these cases were erroneously connected with malaria.

Generally speaking, the *course* of epidemic cerebro-spinal meningitis is *acute.* In grave cases death usually ensues by the end of the first week; more frequently, however, at the end of the second or third week. *Death* results during coma amidst general exhaustion, paralysis of the sphincters, meteorism, and profuse perspiration; towards the end the pulse is feeble, frequently irregular; the temperature is abnormally high or subnormal.

Not infrequently, however, the disease lingers for weeks or months. In such cases death ensues amidst *symptoms of cerebral pressure* from complications, bed-sores or exhaustion, and frequently *extreme* emaciation.

Relapses are not infrequent; and especially during convalescence one is never safe from unpleasant surprises.

Diagnosis.—The *diagnosis* is not difficult, particularly when the disease appears in the course of an epidemic and is accompanied by the cardinal symptoms mentioned above.

Above all, its sudden onset distinguishes this kind of meningitis from the other forms, especially the tubercular. The herpes and the early appearing opisthotonos differentiate it also from the tubercular form. Any possibly existing tubercular focus should be taken into consideration in the diagnosis.

The most decisive test, however, is afforded by *lumbar puncture*, which furnishes a certain as well as indispensable diagnostic expedient.

The fluid withdrawn by lumbar puncture is cloudy, flocculent, and more or less purulent. In the centrifuged sediment the meningococci described above may be detected; they are to be found either in the pus cells or in the intercellular fluid, although occasionally they are so scarce that only a persistent search will reveal them. If no cocci are found, it will be necessary to make cultures on agar or *Loeffler's* blood-serum. Not infrequently, however, we do not get a pure culture, since, as we have already mentioned, it is sometimes a matter of a mixed infection with pneumococci or the ordinary pyogenic bacilli. The cellular elements consists preponderately of polynuclear, less of mononuclear leucocytes. The pressure of the lumbar fluid is more or less increased; not infrequently, especially when a hydrocephalus has already supervened, it is enormously increased (to 500–600 mm. water). The albuminous content is augmented.

With regard to the differential diagnosis, difficulties may arise in distinguishing this disease from *typhoid fever*, particularly when there is typhoid-fever-like rash, high fever, or some stupefaction. In some cases, typhoid may be recognized by its slower onset, meteorism, diarrhœa, the bacilli findings in the stools, and the fluid withdrawn by lumbar puncture, which is nearly clear in typhoid. In epidemic meningitis, excruciating headache with rigidity of the neck predominates; a herpes is a sign rather of meningitis. It must be taken into consideration, however, that in typhoid, meningitic symptoms may manifest themselves, which are caused either by typhoid bacteria (found in the lumbar puncture fluid) or by typhoid toxins. The symptoms are not, indeed, so violent as in meningitis; for which reason they have been designated according to the French practice as meningismus typhosus. (The Widal reaction favors typhoid fever. Leucocytosis, in the acute stage favors meningitis of epidemic type.)

Apoplectiform meningitis is distinguished from cerebral hemorrhages, embolism, and thrombosis, chiefly by the fever and the rigidity of the neck.

In children, a *gastro-intestinal catarrh* may occasionally produce meningitic symptoms, and not infrequently rigidity of the neck and convulsions have been observed with it. However, the condition of the stools, of the sensorium, in some cases opisthotonos, will soon disclose the true nature of the disease.

In *uræmic coma*, fever, herpes, and hyperæsthesia are as a rule absent.

With regard to *serous meningitis*, the cerebro-spinal fluid which in this disease is clear and under high pressure, is decisive; besides, the symptoms in these cases are less violent, the hyperæsthesia and rigidity of the muscles less pronounced.

The differential diagnosis relative to *hysteria* will be discussed below.

Prognosis.—The *prognosis* is always *grave.* The cases in young children and infants which take a rapid course end fatally. According to *Heubner*, about half of those suffering from subacute and subchronic cases escape with life. As is usual, the death rate in the various epidemics fluctuates. *Florand* places recovery at 25–30%; *Netter* at 63%; *Kohts* at 68%; on the average it will be safe to assume a mortality of about 40%.

Experience shows that cases having a violent onset end in an early coma. Those cases beginning more mildly, in which consciousness is affected only later, afford a better prognosis. The latter will then be determined chiefly by the not infrequent complications with other infectious diseases.

Among the *sequelæ*, blindness, deafness, and hydrocephalus furnish the worst prognosis, although *Axenfeld* observed a change for the better even in apparently desperate cases of meningitic ophthalmia, and even hydrocephalus may retrogress after months or years.

The *nervous troubles*, headache, dizziness, tinnitis aurium, neuralgia, occasionally remain for the rest of the patient's life.

Treatment.—It is possible to institute general prophylactic measures by early and complete isolation of all sporadic cases.

Absolute rest in bed, complete silence in his vicinity and the darkening of the sick room are made necessary by the hyperæsthesia of the patient.

From the very first great care should be used that the patient gets sufficient *nourishment.* Since, as a rule, there exists an aversion to food, it will often be difficult to follow out this rule. The most favorable opportunities afforded in the course of the disease should be used to give the patient foods as rich as possible in heat-units. Not infrequently sufficient nourishment can be given the patient only by means of a stomach tube, of which method *Heubner*, for instance, freely avails himself.

In order to influence *the inflammatory processes*, an application of ice bags to the shaven head, sometimes the application of *Chapmann's* or *Leiter's* coils to the head and spinal column is to be recommended. For the same purpose, cool water is repeatedly poured over the neck and back of the patient. With robust individuals, one should consider blood-letting by means of cupping glasses placed along the spinal column, or by the application of leeches in the region of the mastoid process. *Clearing out the bowels*, particularly in cases with a violent onset, by means of calomel (0.2–0.3 gr. several times, for children correspondingly less), derivation to the skin by means of inunctions of mercurial ointment upon the shaven head, painting it with tincture of iodine or with 10% iodoform salve, applying a blister behind the ears to the neck or along the spinal column, the actual cautery along the course of the latter, are all remedies which, at least symptomatically, may be used with success.

Aufrecht, Wohlisch, Heubner and others, have obtained undoubted benefit from *hot baths; Heubner starts* at 95 degrees, increasing one degree every day up to 102 and 104 degrees and even higher. He follows up the bath with a diaphoretic pack. As a rule he gives one bath daily to his patients, but occasionally repeats the procedure on the same day.

When *violent pains* and *high temperature* exist, it will often be difficult to avoid the use of anodynes or antipyretics. The latter, however, should be used with great care to guard against the danger of a collapse.

For marked *jactitation*, convulsions, delirium, one will have to resort to morphine (1/4 to 1/2 gr.) or chloralhydrat (5 to 10 gr.); it is advisable to give the latter in the form of enemata (20 to 30 gr. pro dosi) if no stupefaction exists.

Lately *lumbar puncture* as a therapeutic means has made great headway. A large number of reports have dealt with this method of treatment, but the results are still uncertain. A priori it seems reasonable to permit the pus to flow off, and since lumbar puncture is to be regarded as a rather harmless procedure, its application should always be tried. Naturally, one puncture will not suffice; it should be repeated frequently, daily or every other day, according to the pressure, and 20–50 c. cm. (*Zupnik* advises even 70–90 c. cm.) should be withdrawn each time.

The effects consist of a decrease of the pressure symptoms, of headache, dizziness, and a clearing up of consciousness. Wider experience must determine in those cases which recover under this treatment, if the result be due really to puncture.

Surgically, *trephining* with opening of the dura and a washing out of the subarachnoid space has been recommended, but so far as we know no success has as yet been obtained by this method.

Recently, attempts at a *specific treatment* of epidemic cerebro-spinal meningitis by the use of the *meningococcic serum* have been instituted. *Wassermann* claims to have seen good results follow early injections of 10–20 c. cm. of his serum; favorable reports of the serum treatment have come also from other sources.

So many difficulties present themselves, however, in deciding the curative value of a serum in an epidemic disease, that only after years of observation by impartial critics can an ultimate decision be rendered. (The value of serum treatment is now pretty well established, and Dr. Simon Flexner has done much work proving its great importance and usefulness.)

B. Secondary Purulent Leptomeningitis

(*Meningitis Simplex*)

Under this head we include (a) *consecutive* (*fortgeleitete*), (b) *traumatic*, and (c) *metastatic resp. septic forms* of leptomeningitis.

Even though this classification be founded solely upon the ætiological viewpoint, it must be noted that the disease picture possesses certain characteristic peculiarities, according to the cause inducing the disease, and that it deviates considerably from that of epidemic cerebro-spinal meningitis.

Common to all these secondary forms of meningitis, however, is that each of them may be caused by the *most varied pathogenic excitants*, so that in this respect they are *not ætiologically uniform.*

Infection Excitants.—These comprise nearly all the *pyogenetic excitants* that are known to us to-day.

Thus there have been found in the purulent exudate *streptococci*, and *staphylococci* (chiefly *St. pyogenes aureus*), also *bacilli of the coli group*, the *bacillus pyocyaneus* and the *bacterium lactis ærogenes*, and also the bacilli that are generally regarded as specific or as including specific diseases; thus, in the pathology of meningitis an important rôle is played by *Fraenkel's pneumococcus lanceolatus, and Friedländer's pneumo-bacillus; Pfeiffer's bacillus hæmophilus* (*the bacillus of influenza*) also belongs among the excitants of meningitis, as does the *bacillus of typhoid.* Finally we mention the *meningococcus intracellularis* (*Weichselbaum*) which has been found in many cases of sporadic meningitis. It is doubtful, whether such forms should be classed with the epidemic or with the secondary form of meningitis. But since this micro-organism may be present in the naso-pharynx of healthy persons also, it is not impossible that in diseases of the naso-pharynx the germs might reach the soft membrane of the brain through the lymphatics.

Hence the difference between these forms and epidemic cerebro-spinal meningitis would consist only in the absence of an epidemic character in the former, and since the clinical picture does not seem to differ in any way from that of epidemic cerebro-spinal meningitis, we shall not dwell on these cases any longer in this place.

With striking frequency *Fraenkel's diplococcus* is found to be the generator of meningitis even in the course of epidemics, for which reason some have refused to give epidemic cerebro-spinal meningitis a specific significance or *to admit its ætiological uniformity* (*Leyden, Stadelmann and others*). But so far as we know, no epidemic entirely due to Fraenkel's diplococcus has been recognized, while in sporadic cases, this excitant has been found comparatively often. We therefore agree with *Heubner*, who regards only those cases as epidemic cerebro-spinal meningitis which have been induced by the meningococcus intracellularis.

It has been remarked that at certain times cases of pneumococcic meningitis may become so frequent as to resemble an epidemic and in such cases it must be remembered that pneumonia likewise often assumes an epidemic character.

Mixed infections are frequent; in such cases the exudate frequently contains besides the specific bacteria, the ordinary pyogenic excitants, such as staphylococci and streptococci.

Experimental methods have been successful in inducing a meningitis by injection of pneumococci into the subarachnoid space. *Fraenkel's diplococcus* has a thick and a thin end; they lie in pairs, with the thicker end together, enclosed in a capsule (cf. Fig. 159, p. 572). The staining is done with aniline colors; the coccus becoming stained while the uncolored capsule surrounds it as a light space. Should the microscopic staining test not succeed, the decision may be reached by experiment on animals: injected into rabbits and mice, it produces a general infection.

Whether the *nature of the excitant of the infection* has a decisive influence upon the disease picture, has not yet been definitely ascertained. Of greater importance, as was said before, is the manner of the infection.

(a) Consecutive Meningitis

This disease owes its origin to the migration of infectious or purulent matter into the subarachnoid space from neighboring organs. There are three ways by which their entrance is effected: First, by the *lymphatic vessels;* secondly by the *nerve-sheaths*, and thirdly, by *direct contact* of the cerebral membranes with the focus of infection by continuity. The *blood-vessels* seem to play little or no part in this form.

As conductors along the lymphatic pathways, the cavities of the skull, which give shelter to the organs of sense, must be considered preeminent. These cavities are evidently connected with the subarachnoidal space by numerous delicate lymphatics. Transference through the nerve sheaths may be rendered possible either by the cranial nerves or the spinal nerves through the intervertebral foramina.

In some cases, it is impossible to determine which path the excitant of the infection has taken.

Probably the most important ætiological rôle is played by *diseases of the skull.*

Foremost among them are:

(a) Those of the petrous portion of the temporal bone, resp. of the *organ of hearing.* As a rule it is otitis media which has supervened upon a caries of the petrous bone, which perforates the thin roof of the tympanic cavity; the inflammation thereby proceeds directly to the membranes of the brain. The cause of caries in the petrous bone may be found in a cholesteatoma as well as in a tubercular or a syphilitic affection. From the *mastoid process* a purulent infection may also proceed to the meninges.

But it is not only by continuity that an affection of the meninges is possible in otitis purulenta. Even if the petrous bone is not perforated, the micro-organisms may reach the pia mater by the way of the *sheath of the facial or auditory nerves*, as well as by means of the vessels running through the petrosquamosal fissure.

Not infrequently the connecting link is formed by a *thrombosis in a* *neighboring sinus*, as in the superficial petrosal sinus, the transverse sinus, and the cavernous sinus. Thrombophlebitis leads first to suppuration in the sinus, and secondly to a meningeal infection.

Recently, however, it has been repeatedly stated by the specialists in children's diseases, that in infants the clinical observations contradict the view that otitis media purulenta without a labyrinthine disease leads to meningitis. Thus, e. g., *Thiemich* is of the opinion that in severe cases a *secondary infection* of the middle ear is to be assumed. The writer can agree with this view only when anatomically a connection between meningitis and the middle ear can be proved, and when the ear disease has preceded the meningitic symptoms.

(b) Next to the ear, *the nose and its accessory cavities*, the sphenoidal, ethmoidal and frontal sinuses are considered as important sources of infection of the meninges.

I wish to call especial attention to these sources of infection, as it seems to me that many cases of meningitis of mysterious origin may be explained by them. Of course, the assistance of a specialist is required to determine accurately *ethmoidal and sphenoidal affections*, and it is therefore advisable to consult a nose specialist in all doubtful cases.

I myself have treated two cases in which the supposedly rare source of the infection was found in the *sphenoidal sinus*, and both of these were cured by providing better means for the pus to flow off by the use of cocain-tampons and mechanical enlargement of the sphenoidal opening.

These cases encourage future proceedings along the same line. Painstaking care, must, however, be always given to formulating the diagnosis, and, of course, to finding the source of the infection.

(c) A further source of infection may be a *purulent focus located in an orbital cavity*. Thus, secondary meningitis has been seen to set in after *panophthalmitis*.

(d) We further mention *cerebral abscesses*, which may reach the meninges by means of peripheral dissemination; and *extra-dural abscesses* which usually emanate from *suppurating hæmatomata*.

(e) According to *Heubner*, infants with *syphilitic* rhinitis, and those *with whooping cough* are often affected with purulent meningitis.

(f) Occasionally cases of *erysipelas of the head*, *boils* on the neck and scalp, or cases of *purulent infections of the scalp*, and *parotitis*, may by the dissemination of the excitants of inflammation develop meningitis.

(g) Lastly, those cases of *ascending leptomeningitis* in which the primary purulent focus is located near the vertebral column, as *bed-sores*, *caries of vertebræ and of the ribs* belong here. Perhaps also those cases should be grouped here in which meningitis follows *pleuritis*. The sheaths of the *intercostal nerves*, in such cases, are regarded as the pathway along which the disease travels.

(b) TRAUMATIC MENINGITIS

This form frequently follows *fracture of the skull* with open wounds. The micro-organism enters from the outside through the dura; sometimes by means of a thrombosis in a sinus.

Meningitis has also been seen to arise in connection with fractures of the base without open wounds. In such cases, the excitant probably penetrates into the cranium through the external auditory meatus, or the nasal cavity.

The question whether meningitis may originate *without lesion of the soft parts and of the cranial bones*, can be answered in the affirmative, on the ground of numerous experiences (for instance, in tubercular and influenzal meningitis).

(c) METASTATIC OR SEPTIC MENINGITIS

is most frequently observed in the course of *typhoid*, *influenza*, *articular rheumatism*, ulcerative *endocarditis*, sepsis, and especially in the course of, or following *pneumonia*. *Acute eruptive diseases*, as scarlet fever and small-pox, are occasionally associated with meningitis. It is, however, often very difficult to decide in such cases whether the meningitis developed metastatically, or whether it is only a partial symptom of a blood infection.

The **pathological anatomy** of secondary meningitis, in its essential points, coincides with that of the epidemic form. But it should be noted that in the consecutive and traumatic form, the *most marked changes*, and *accumulations of exudate*, are situated *at the place of the neighboring primary focus;* from this focus the purulent, sero-purulent, or fibrino-purulent exudation extends primarily along the blood-vessels (veins) over the surface of the brain. In the secondary form, quite as in epidemic cerebro-spinal meningitis, the *most marked changes* are seen on the *convexity*, while the base is comparatively less seriously affected. It is indeed true that, as we have already mentioned, many exceptions occur particularly in the consecutive and traumatic forms; in *otitic meningitis*, we correspondingly find the *largest mass of exudate at the base;* and while we find the spinal meninges comparatively free, in secondary meningitis (in contradistinction to epidemic meningitis), the ascending form now proves an exception, for in this variety the inflammation extends largely and mainly along the under surface of the spinal pia.

In the secondary form, just as in epidemic meningitis, the *substance of the brain participates in the inflammation;* hemorrhages, inflammatory foci, small purulent foci, etc., are the usual findings in the cerebral cortex.

The **clinical picture** is distinguished from that of the epidemic form principally by the fact that the symptoms of meningitis intrench upon the picture of the causative disease. Not infrequently, it is very difficult to differentiate the symptoms of meningitis from the symptom picture of the fundamental disease, as, for instance, in *typhoid* which so frequently simulates the picture of meningitis.

The *septic* form in most cases has exactly the same clinical picture as the epidemic variety, so that we refer the reader to the latter. Likewise, the consecutive and traumatic forms may occasionally assume the same character.

In *infants and small children*, the course is more violent. Within a few hours the clinical picture reaches its climax with vomiting, high fever, convulsions and respiratory acceleration. "Soon the breathing assumes a peculiar moaning sound during expiration, forming the only expression of the pain from which the infant in all probability suffers," says *Heubner*. The facial expression becomes distorted, consciousness becomes clouded and lost. Signs of intra-cranial pressure supervene; the pulse becomes slower and the fontanelles bulge. Within a few days (2–5) death ensues.

In *adults* the disease may also take a *rapid* course. It makes its advent frequently with a *chill*, perhaps after otitis media has preceded it by some days; the temperature rapidly rises to 103 degrees; headaches come into the foreground and now dominate the clinical picture; consciousness is clouded, and stupor alternates with violent delirium, which just as rapidly may change into deeper sopor and coma. When consciousness is clear, vertigo manifests itself. There may now be added paralyses of cranial nerves, pupillary inequality, or possibly rigidity of the neck. *Optic neuritis* may be found. Occasionally convulsions, followed by paralyses of the extremities occur as well. General hyperæsthesia, vaso-motor excitability, at the beginning an exaggeration of the tendon reflexes play a certain part in this form too. *Kernig's* symptom of which mention has been made (cf. p. 559 and Fig. 161, p. 580) is almost invariably present.

The *temperature curve* takes an extremely irregular course, the pulse is usually quickened and irregular; a slow pulse is observed less often in these cases than in the epidemic form. Nor do *rigidity of the neck* or *symptoms of cerebral pressure come to the foreground as much as in the latter*.

The **course** is usually rapid and unfavorable. Death ensues in about a week to a week and a half.

It may be regarded as characteristic of the *consecutive* and *traumatic* forms that the *symptoms indicate in a measure the location of the primary focus*. In meningitis emanating from a disease in or about the petrous bone, the affection frequently starts with a paresis of the facial nerve or clonic convulsions in the area of that nerve; and in general *the picture in these cases more closely resembles that of basal meningitis*. Nuchal rigidity is also more frequent in these cases. In those cases emanating from the *nose*, the basal symptoms are less pronounced and rigidity of the neck sets in late; on the other hand, in patients whose senses are less benumbed, more *disturbances* manifest themselves *in the psychic domain*. Besides the headache is of a more frontal character. In *traumatic* diseases near the motor spheres, symptoms of irritation of the motor regions of the cortex—convulsions of the contralateral side—may be the first symptoms of meningitis to manifest themselves.

From what has been said, it will be apparent that the clinical picture in cases of consecutive and traumatic meningitis corresponds more to a focal disease than to one of a diffuse character, and that for this reason one should always be on the lookout in such anomalous cases for a possibly primary affection in a neighboring region.

The **prognosis** is usually *unfavorable*, more unfavorable than that of epidemic cerebro-spinal meningitis. Recently, indeed, more and more cases of secondary meningitis have been known to recover. I myself know of a number of cases that recovered, in which the purulent nature of the meningitis had been determined by the lumbar puncture. *Pneumococcic meningitis* furnishes a particularly bad prognosis; I had one case in which pleuritis and endocarditis set in in connection with a pneumonia of the upper left lobe, which resulted in death. Pneumococci were found in the purulent fluid withdrawn by lumbar puncture as well as in microscopic preparation of the vegetations upon the valves of the heart. It was, therefore, a typical case of pneumococcic sepsis. Another case of pneumococcic meningitis, however, took a favorable course. I add here illustrations of the preparations of fluid withdrawn by lumbar puncture in the various stages of the disease.

A relatively *favorable* prognosis is furnished, of course, by those cases in which *an infection of the blood has not yet taken place*, consequently the consecutive and traumatic forms.

Even when the danger to life is past, similar conditions to those following epidemic meningitis may remain. Hydrocephalus, however, is not as frequent. In the second case mentioned, that of empyema in the sphenoidal sinus with purulent meningitis, psychic disorders persisted for about half a year. The patient to whom I alluded, with the pneumococcic meningitis, recovered exceedingly slowly, and had to contend with very troublesome dreams, headache, vertigo, and emaciation.

Differential Diagnosis.—Relative to the differential diagnosis, we refer to the discussion in the chapters on epidemic and tubercular meningitis. Occasionally, secondary meningitis may present more difficulties in formulating a diagnosis, than the epidemic variety, particularly when it occurs during an epidemic.

The secondary form can usually be distinguished from the *epidemic* variety by its violent course, its focal character, and the absence of opisthotonos; besides, herpes occurs much more infrequently in the secondary than in the epidemic form. If the primary focus can not be found, a decision will have to be reached by means of lumbar puncture.

Typhoid and pneumonia (particularly pneumonia of the upper lobes) may sometimes show symptoms resembling meningitis, especially in cases where headache, some stupefaction, and stiffness of the neck predominate; but the temperature, the absence of symptoms of paralyses, the positive *Widal* reaction, and the bacilli findings in the stools all indicate the former. In cases in which it is particularly difficult to reach a decision, lumbar puncture will be decisive.

Nor will *pneumonic* affections (presumably it will be a matter of central ones) long escape the notice of a skilful examiner.

There are cases with meningitic symptoms which lead to death, though no meningitic changes can be found if an autopsy be had. Hence these cases have been designated as *meningism.* In the course of infectious diseases, such as typhoid fever, e. g., one has opportunity to observe such cases.

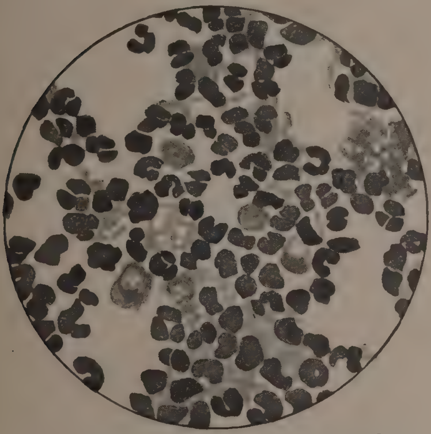

FIG. 157.—Purulent (pneumococcus) meningitis (Nissl's method). Polynuclear leucocytosis. Fluid withdrawn by puncture purulent. Leitz Okl. Imm. 1/12 Ok. 3 (Author's observation).

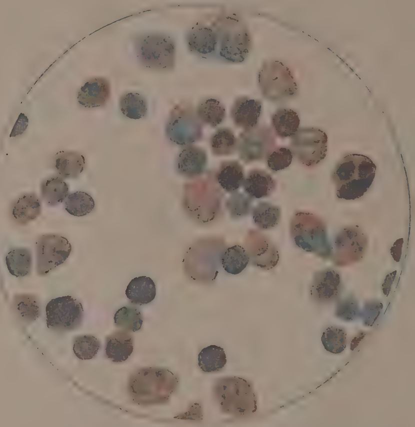

FIG. 158.—The same case. Fluid withdrawn by puncture. (Stained according to May-Grunwald.)

Autopsy reveals no meningitis; at the most, some hyperæmia. In such cases, it is evidently a matter of toxic lesions of the membranes of the brain, which can not be detected anatomically.

Finally, we call attention to *pseudo-meningitis*, which depends upon an hysterical foundation. *Hysteria* may imitate typically every form of meningitis, a fact to which *French* authors first called attention. Nuchal rigidity, vomiting, chills, fever, incontinence, exaggerated reflexes, strabismus —all these may simulate meningitis very closely. Yet the careful observer will, nevertheless, discover hysterical traits in the clinical picture which will soon lead him to doubt the organic nature of the disease; and if not, hysterical stigmata, the fact that an increase in temperature is lacking—of which one becomes convinced by taking it personally—will soon disclose the true nature of the disease. In 1901 I published a report of a striking case of psychogenous pseudo-meningitis in the "Zeitschrift fur Nervenheilkunde." In that case, detection was accomplished by means of hypnotism.

I have constantly mentioned *lumbar puncture* as an important diagnostic adjuvant. Indeed, in many cases it seems to me to be the only certain means of recognizing the form of meningitis, and should therefore be used in every case showing meningitic symptoms. It is often surprising to find an abnormally turbid and purulent fluid while but few cerebral symptoms exist. There are, undoubtedly, also cases of secondary meningitis in which the purulent nature of the fluid is still lacking; the microscopic examination of the centrifuged sediment reveals, however, the micro-organisms. If this is not the case, it will be necessary to make cultures, or to have recourse to animal experimentation. In such instances it must always be remembered, that the meningitis often depends upon a mixed infection, or that the primary specific infection may be complicated by a secondary germ.

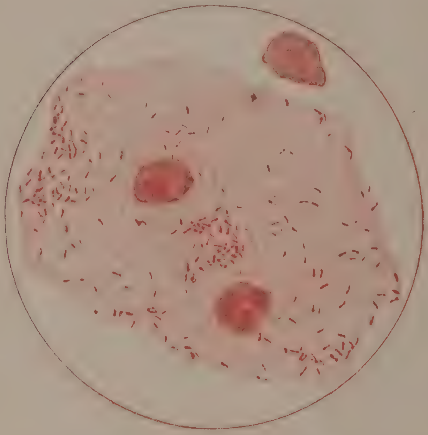

FIG. 159.—The same case. Spread preparation of the centrifuged fluid with numerous pneumococci. (Fuchsin staining.)

The *cells* in purulent meningitis consist principally of polynuclear leucocytes, and a smaller number of lymphocytes.

When puncture is resorted to repeatedly, it will often be easy to follow the *progress of the disease*. Even to the naked eye the cerebro-spinal fluid may indicate an amelioration by becoming clearer.

The microscopic preparations made with careful uniform technic also reveal the progress of the disease. These usually show very clearly how the wealth of cells in later punctures has decreased and how the polynuclear leucocytes have entirely disappeared, and how few lymphocytes have remained.

We can not forbear to mention, however, that the purulent meningeal fluid is occasionally *sterile*, i. e., the micro-organisms can not be found either under a microscope or on the culture plate.

Therapy.—The general treatment coincides with that of epidemic meningitis.

We call attention to the necessity upon *prophylactic* grounds of a careful aseptic treatment of all wounds in the skull, suppurations, etc. The fact that meningitis may supervene in operations upon the nose and the ear make the utmost care and absolute cleanliness imperative.

How important it is to find the primary focus is indicated by the above mentioned cases of sphenoidal disease. An incipient meningitis may be brought to a standstill by aspiration or trepanation of the mastoid process. In cases in which a preceding trauma or the symptoms point to a focal disease, an attempt should be made to remove the accumulated exudate by means of trepanation. A number of cases have been saved by timely operative interference. When the pus is already diffused, however, one can not expect much even from an operation. In some cases, *ventricle puncture* has been the means of recovery (*Beck*).

Authorities do not as yet agree with regard to the usefulness of *lumbar puncture*. Undoubtedly amelioration may be and has been obtained by it and even recoveries have been ascribed to it by conscientious observers. I have had one case in which I do not hesitate to ascribe the recovery to lumbar puncture.

This case, and similar ones described in the literature, at any rate encourage the use of lumbar puncture in every case of meningitis, and its repetition if possible every second day. The amount to be withdrawn depends upon the pressure of the flowing fluid. Where marked symptoms of brain pressure exist, up to 100 c. cm. may be withdrawn.

Quincke recommends *slitting the dura*, but we have no precedents for this as yet. Nor can one as yet properly judge the *washing out of the subarachnoid after trephining* the spinal column.

C. Tubercular Meningitis

Ætiology.—Tubercular meningitis appears to be always a *secondary* disease, occasioned by the tubercle bacilli migrating to the pia mater. *The primary disease* may have its seat in any of those organs of the body in which chronic tubercular foci are wont to be situated.

Most frequently the disease seems to manifest itself in connection with the *pulmonary* form of tuberculosis. This is especially the case in advanced and terminal cases, particularly in adults, though not infrequently the lung affection is very trifling or has seemingly been recovered from.

Next in importance to the lungs are tubercular *lymphatic glands*, such as mesenteric glands, but above all, the bronchio-tracheal glands, the glands of the neck, and the deep-seated parapharyngeal glands. The latter seem to play the main part in infantile tubercular meningitis.

It is claimed that not infrequently tuberculosis of *serous membranes*, especially of the pleura is the origin of a tubercular meningitis.

Somewhat more infrequently one finds the source of a meningeal disease in a tubercular *bone or joint affection;* and *genital tuberculosis* (testicular) also plays an ætiological part which should not be underestimated.

It happens occasionally that a postmortem examination at first fails to reveal the primary focus, and that only after a thorough search of the entire body (spinal cord, cavities of the skull, sphenoidal sinus, inner ear, larger lymphatic channels, *ductus thoracicus*) the starting point of the disease can be found. An undoubted case of primary tubercular meningitis is, however, not known as yet.

The question *in what way* and under what *conditions* the secondary infection of the meninges takes place, has not met as yet with a satisfactory solution.

In the majority of cases it may be taken for granted, that an invasion of tubercular matter containing bacilli into the *blood stream* has taken place; this is proved by the frequent finding of a synchronous miliary affection of the other organs of the body, by synchronous *general miliary tuberculosis*, by the embolic spreading of tuberculosis into the meninges and finally by the frequent cases in which success has attended efforts to find the place of invasion in the vessel.

The invasion may take place either into the venous or into the arterial streams, for instance, into the pulmonary veins.

Another means of infection is by *direct transmission* from an organ vicinal to the meninges; thus a tubercular affection of the *middle ear*, *of the sphenoid bone*, *of the base or vault of the cranium*, *of the orbit*, *and also solitary tubercles* and tubercular *abscesses* of the brain have been followed by a transmission of the tubercular processes to the meninges. Even if in such cases at first, it is a matter only of circumscribed inflammation, the cerebro-spinal fluid may easily cause an inundation of the meninges with matter containing bacilli.

That the *lymphatics* also can transport the tuberculous matter to the meninges, is beyond doubt. This is shown by the frequent absence of miliary tubercular processes in the other organs, the comparatively frequent exclusive appearance of meningitis as a concomitant of tuberculous affections of the tracheal, pharyngeal and cervical glands. *Strümpell* surmises that the infectious matter passes through the *lymphatic channel of the nerves* (for instance the intercostal nerves) first to the subarachnoid sac of the spinal cord and thence farther upward to the base of the brain.

Not infrequently accidental *determining causes* play an important part in the development of the disease; many cases have been reported in which it followed immediately upon *cranial traumatism.* Tuberculous meningitis in infants follows with surprising frequency *infectious diseases* especially *measles and whooping cough*, diseases which evidently, as a rule make the patients more susceptible to infection with tuberculosis.

It has been noted that sometimes many cases appear in the same neighborhood and hence some writers even speak of an *epidemic character* in the disease. It is generally accepted that this grouping of cases most frequently occurs in the spring.

When one speaks, relative to the pathology of tubercular meningitis of a *predisposition*, one has only in view that relating to the *infantile men-*

inges. It is certainly odd and can not be explained by the foregoing factors that tubercular meningitis shows an outspoken predilection for *early or the earliest childhood.* Infants in the first half of the first year are rarely stricken with the disease, it appears most frequently in the second half of the first and in the second year and decreases in frequency until the sixth year and during the school years; then an increase in the appearance of the disease is to be noted from the 18th to the 35th year, while in later periods of life the disease occurs but rarely.

Pathological Anatomy.—The *dura* is quite tense, covered with small hemorrhages; at the base it frequently is covered with little miliary tubercles.

After removal of the dura flattening of the convolutions and a filling up of the sulci upon the convexity attract notice; the whole brain, especially the lateral portions, when palpated create an impression of being filled to overflowing on account of the marked fulness of the lateral ventricles.

At the *base of the brain* we recognize at once the two characteristic pathologic features, the *tubercles* and the signs of *inflammation and exudation.* The latter most striking phenomena consist of a more or less fluid, frequently gelatinous, jelly-like exudate of a whitish, yellowish or greenish-yellow color which fills the subarachnoid spaces. This exudate is most pronounced in the region of the chiasm and between it and the pons at the lower surface of the medulla oblongata. But the exudate also surrounds the larger vessels, fills the cavities and the sinuses and follows the nerve trunks.

If one opens the arachnoidea and carefully withdraws the exudate, the somewhat clouded pia becomes visible, covered with a large number of the *very smallest submiliary and miliary, opaque, whitish tubercle-nodules.* These small nodules frequently can be noticed only when one removes the pia from the cerebral cortex and permits a light to strike it; but in other cases they are so numerous and set together so densely that the pia feels rough and is no longer permeable to rays of light. The greatest accumulation of the nodules is always to be found in the neighborhood of the vessels; they follow the vessels from the base into the finest ramifications, accompany them into the smallest fissures and enter with the *plexus chorioideus* into the ventricles. A more thorough examination shows that they mostly lie *perivascularly* in the tissue of the lymphatic vessels; but moniliform thickenings of the vessels prove also that the wall of the vessel itself is affected or that the tubercular matter obstructs the lumen.

The number of the nodules decreases distally from the base and at the convexity one may no longer be able to discern any with the naked eye.

The *membranes of the spinal cord*, especially the posterior half, show the same changes.

Very frequently *inflammatory and softening processes* are continued from the pia *into the cerebral substance;* in the basal ganglia especially deep-reaching encephalitic changes have been noticed.

In rare cases more circumscribed aggregations of tubercular matter occur in the cortex, or large tubercles, tuberculous plaques, which cause symptoms of tumor according to their position (pons, cerebral, peduncles).

The *cerebral cavities* are almost without exception enlarged by the accumulation of an almost clear fluid. This fluid originates in the plexus, is inflammatory and shows therefore a large percentage of albumen. This hydrocephalus was known in former times as a symptom of tubercular meningitis and hence the disease was designated as *acute hydrocephalus of the cerebral cavities.* Later on, when speaking of lumbar puncture, we shall deal with the condition of the cerebro-spinal fluid.

There are numerous *deviations* from the pathologic-anatomical picture thus far sketched. The extraordinary diversity in the intensity of the inflammatory phenomena deserves especial attention. The exudate may be reduced to a minimum, the base may seem to be entirely free and only a *hydrocephalus*, which at first might be taken for a nonspecific one be present. In other cases the condition of the exudate deviates from the average picture; it is sometimes quite clear, sometimes more purulent, so that the clinical picture of a purulent meningitis is simulated. In still other cases the tubercular nodules can not be seen with the naked eye or only after a very painstaking search one may detect some at predilection spots, for instance, at *places where small vessels separate* and then only in small number. It may also happen that the entire process takes place more at the convexity than at the base, or, it may be restricted to quite circumscribed vascular areas, as to the artery of the fossa Sylvii on *one* side only.

As a general rule the cerebral membranes are principally affected, though in rare cases the process is restricted almost entirely to the membranes of the *spinal cord.*

The *substance of the brain and spinal cord*, too, participates in the disease in a very variable degree. As mentioned before, inflammatory and softening processes of the cerebral cortex occur as a rule, and in the spinal cord, too, one has remarked (*Schultze*) far-reaching changes in the form of an acute interstitial myelitis. The spinal ganglia are also frequently affected.

But in any case, whether the changes be quite atypical or quite insignificant, it will always be possible to prove that *tubercle bacilli* are at the *root of the disease* and to find them not only in the tubercular nodules but also in the exudate.

Clinical Picture.—From the foregoing statements it may be seen that tubercular meningitis must show a very *variable complex of symptoms.* Indeed, the clinical picture in individual cases is frequently so entirely unlike that one is rather inclined to assume the existence of quite different diseases. Frequently diagnosis is possible only by taking into consideration the entire impression of the clinical picture.

Symptomatology.—The *initial symptoms* of the disease differ according to the seat and the extension of the primary tuberculosis. If it be a matter of a *latent tuberculosis* and an invasion of a vessel takes place, then the first symptoms of the disease may consist of *chills* suddenly appearing in full health. The initial local symptoms may be dyspnœa, pleural or gastric pains a "stitch" in the side, or headache—or, to be brief, the symptoms may appear in any organ affected by the miliary dissemination of the tubercular bacilli. Besides the symptoms of general miliary tuberculosis, those of meningitis make their appearance.

In cases, however, in which real meningitic symptoms occur which can not be regarded as part-phenomena of the general miliary tuberculosis, *prodromal phenomena* frequently precede the characteristic symptoms and may last for weeks. Whether the prodromes may be traced to an affection of the meninges, must be left undecided; but many factors make for the opinion that they are to be traced to the flaring up of the primary focus in the bronchial glands, cervical glands, etc.; at any rate, they are usually followed by a meningitis proper. This prodromal stage is especially pronounced in *infants*, especially in cases in which they seemingly have been perfectly well before. They surprise one through their *change of manner:* jovial, jolly children become at once peevish, do not care to play any more, show certain oddities, are at one time more apathetic, at another more irritable and ill-behaved; *the whole character is changed.* At the same time they lose their appetite and become pale and scrawny. Sleep is disturbed; they talk during sleep, fall into delirium and toss about uneasily. An *objective examination* does not show anything positive with the exception, perchance, of an increase or decrease in the normal temperature. Occasionally, without any apparent cause, vomiting appears; upon questioning one learns that transient pains in the occiput or a slight rigidity of the neck is complained of. Abdominal pains in the region of the appendix seem to indicate that the affection has its seat in the digestive canal. As these cases not infrequently are accompanied by constipation, there is further cause for this assumption and the whole treatment consists then in the regulation of the stool. These indefinite symptoms may last for weeks until alarming symptoms set in to indicate a cerebral affection.

In other cases the onset of the meningitis is preceded for weeks by severe *nervous symptoms* such as convulsions, fainting spells, headache, restlessness, irritability; it is likely that in these cases the anatomic precursors of the meningitis consist in circumscribed tubercular foci on the surface of the brain.

In adults, too, though less frequently, one may observe *a prodromal stage of the real disease extending for a longer period;* changes of character and temperament, disorderliness, uncleanliness, dreamy conditions combined with headache and a general feeling of being sick without being able to localize the affection, nausea, lack of appetite, emaciation, and enduring periods of fever. One case that I observed myself (and which did not change into miliary tuberculosis) presented such phenomena for a period of three weeks, during which period not the slightest indication of meningitis or tuberculosis could be observed.

The real *onset of the meningeal affection* may take place under the most varied symptoms: now it occurs in an explosive way which in one day enables one to make a safe diagnosis—now the transition is almost imperceptible. In the latter case, phenomena of an abnormal irritability of the central apparatus set in by degrees; the senses become supersensitive to slight irritations; light, bright colors, noise, become disagreeable; a touch is felt as pain; the nerves of smell and taste become supersensitive. Phenomena of *motor irritation*, too, follow: slight twitchings of muscles, gritting of the teeth, involuntary grimaces, yawning and motor restlessness in bed.

The *vaso-motors*, likewise, show an increase in irritability; flaming redness alternates with marked pallor, circumscribed red spots are observed upon the skin, and a slight cutaneous stimulation produces hyperæmia of the vessels.

Little by little transient *convulsions* alternating with palsies and disturbances of consciousness are added to the clinical picture.

It has been attempted to divide the symptom complex of meningitis into *different stages* and thus to distinguish between a febrile, an apyretic and a lethal stage, or to mark off a stage of irritation from one of pressure and relaxation (*Traube*).

As the changes in the frequency of the pulse in many cases show a certain regularity, the disease has been divided into three stages, namely, before, during and after slowing of the pulse (*Whytt, Kohts*).

In my opinion such division in three stages is practically not feasible, as the course of the disease is too manifold and the symptomatology too irregular; it is *precisely the atypical course that seems to me to be the main characteristic of the disease.*

Nevertheless, if one looks at a larger number of cases, it is not difficult to recognize, that in the final stage of the disease *paralytic symptoms* predominate, while in the initial stage *irritation phenomena* stand in the foreground of the clinical picture. These, as mentioned before, are the first safe criteria of a meningeal affection. It is true that in some rare cases the disease is ushered in so violently, that one may think at first of a case of epilepsy; indeed, the disease may set in with an *epileptic attack;* the paroxysms are mostly confined to restricted parts of the body, half of the body, one arm, one leg, the facial nerves and simulate in this way cortical epilepsy; in other cases slight twitchings of single muscles may be noted. Not rarely the convulsions are followed by a transient paralysis of the muscular group concerned. One of the most frequent phenomena, however, is a peculiar *rigidity of the musculature*, especially of the back of the neck, but also of the extremities and of the abdomen. If the patient in this stage of the disease still walks about, the most conspicuous feature is the *stiff carriage of the body;* the lumbar portion of the vertebral column shows a lordosis, the head is rigidly drawn backwards. The abdomen is scaphoid, its musculature spasmodically contracted. In the face one sees frequently a tetanic contracture of the one side combined with a paresis on the other. Another phenomenon, *trismus*, a tetanic contracture of the muscles of the jaws, is likewise frequently observed. Passive straightening of the contractures is rarely possible without severe pain. *Nuchal rigidity* is frequently so severe that it becomes quite impossible to move the head forward; if one attempts this movement the whole trunk of the patient is lifted stiffly.

Rapid change in muscular tonicity is furthermore very characteristic; a contracture may yield within an hour to a quite normal tonus or even to a flaccid paralysis and one sees frequently in the course of the disease one group of muscles change several times from a state of irritation to that of paralysis.

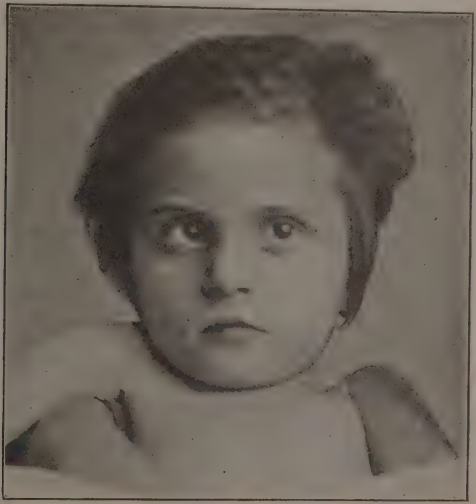

FIG. 160.—Meningitis tuberculosa. Paralysis of the abducens.

The *tendon reflexes*, too, undergo the same variations; at the time of the predominance of irritation symptoms the patellar reflex as well as the Achilles tendon reflex is active and even exaggerated; not infrequently a positive *Babinski* reflex may be observed. *In the terminal stage the patellar reflex is frequently lost;* it is very characteristic that the bilateral *reflexes* are *not uniform* on both sides, that the reflex on one side may be exaggerated, while that on the other side may be normal or entirely lost. It happens also, and not as a rare exception, that the tendon reflexes remain normal until the terminal stage. The reflexes of the upper extremities much more rarely show deviations from the normal than those of the leg. Disturbance of the *sensibility* is rarely present save in the form of *hyperæsthesias* of the skin, musculature, bones and joints which are regular symptoms of the disease. An increase in the vaso-motor irritability of the skin is likewise rarely lacking.

Regarding the nerves—disorders of function of the *cerebral nerves* play the most important part; frequently they first enable us to make a correct diagnosis and they may be observed in every stage of the disease even the prodromal.

As we have seen in the section devoted to pathological anatomy, the exudative process is mostly localized between pons and chiasm, where the oculomotor and abducens are frequently found quite imbedded in the mass of exudate; therefore it is easily explicable that it is principally these nerves whose function becomes impaired. As a matter of fact, frequently one of the first and most lasting symptoms of the disease is unilateral or even bilateral *ptosis*. The pupils rarely remain normal; they show now miosis, now mydriasis, or a difference in the width; the reaction is sluggish, strong *irritation* of the *skin* produces sometimes reflex-dilatation of the pupils; in the terminal stage they become large and do not react at all.

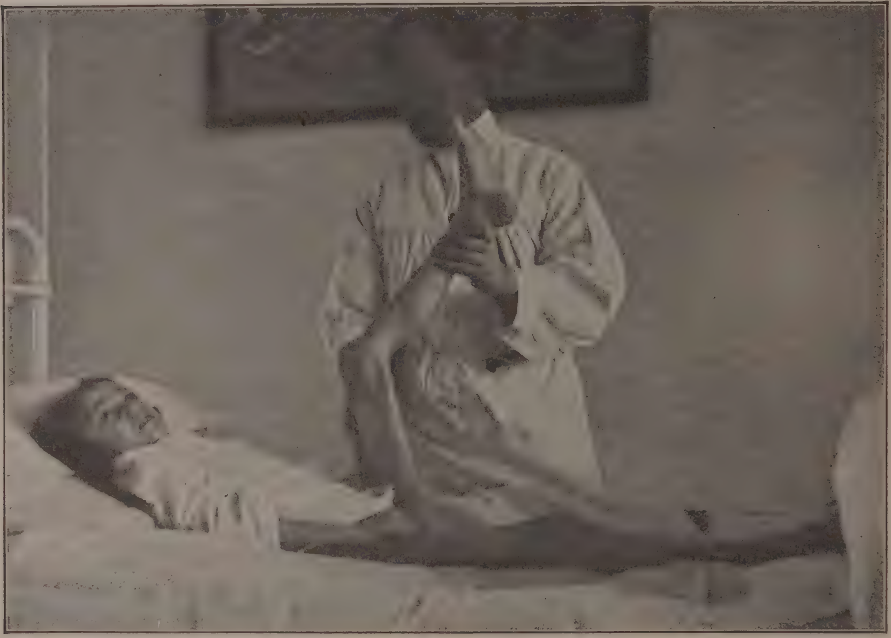

FIG. 161.—Tuberculous meningitis. Test of the Kernig symptom.

Lack of parallelism of the globes, inner or outer deviations and squinting belong to the symptom-complex (see Fig. 160); sometimes one may observe in the eye muscles irritation phenomena, e. g., the eyeballs deviate involuntarily or show nystagmic twitchings.

Ophthalmoscopic examination shows very frequently pathological changes in the fundus, such as hyperæmia, discoloration of the disc, choked disc, neuritis optica. The occurrence of chorioidal tubercles (that can be detected ophthalmoscopically) seems to be extraordinarily rare. In my experience chorioidal tubercles could be found only once by the eye specialists in about fifty cases of tubercular meningitis that I have observed in the last four years.

I have already spoken concerning involvement of the facial; in rare cases a paralysis of the hypoglossal, too, has been observed. The *spinal nerves* have been found frequently to be tender to pressure; a symptom that is only rarely lacking is the so-called *Kernig's* sign, that is, the inability to flex to a right angle the leg extended at the hip-joint when lying upon the back. (See Fig. 161, p. 580.)

The *temperature of the body* runs a very unlike course in the various cases. It depends frequently on the fundamental disease, on the concomitant tuberculosis of the lungs or glands or on a synchronously existing *miliary tuberculosis*. So we see now a typically intermittent curve, now one that remains regular, now a hectic fever, now an atypical course of feverish symptoms, just as occurs in moderately developed pulmonary phthisis.

If it be a case, however, of *latent tuberculosis* which hitherto ran its course without fever, if therefore, the temperature is solely dependent upon the meningeal affection, then we usually observe *moderate fever*, which rarely reaches a high degree or shows features characteristic for the disease; now there is a higher temperature in the morning, now in the evening; not rarely we see the temperature fall below the normal only to ascend sharply but irregularly; it rarely goes above 102 and is frequently below 102, but as a whole shows no regularity in any direction.

In *children* not infrequently *a fall of the temperature* to normal is observed *in the terminal stage of the disease*, followed in the agonal stage by a marked increase and after death by hyperpyrexia.

The *pulse* also shows frequent changes; in the initial stage it is usually accelerated but regular and corresponds more or less to the degree of the fever; in the terminal stage of the disease, when the paralytic phenomena push to the fore, it varies frequently to a remarkable degree. Frequently sudden *slowing* takes place, it falls to the normal or even below it and becomes at the same time weak and irregular, unequal and intermittent; slow beats are followed by very rapid ones. This *constant change of the pulse* beat is always an unfavorable symptom and indicates an increase in the cerebral pressure. With the agonal rise in the temperature the pulse rate also increases frequently to 150 beats and even more.

The *heart* too is concerned with the fluctuations of the pulse; especially striking are the rhythmic disturbances.

The other *internal organs* only rarely show marked changes. Occasionally an enlarged spleen may be observed. *Sluggishness of the bowels* continues frequently until death; sometimes spontaneous exanthemata and erythemata appear on the skin.

The *terminal stage* is, as we have said, characterized by the phenomena of paralysis. The restlessness, the symptoms of irritation, delirious states, yield more and more to a general debility. Transient or enduring paralyses replace them; hemiplegias, monoplegias and paraplegias appear and aphasic disturbances, also, are added to the clinical picture in this stage. The sphincters are paralyzed. The patient can not take any more nourishment because deglutition is no longer possible. The respiration, which before was accelerated, becomes superficial, irregular, and acquires the type of the Cheyne-Stokes respiration. Consciousness vanishes by degrees until the patient falls into a deep coma, from which there is no more awaking.

The anatomic findings make possible a correct interpretation of the cause of most of the symptoms. The symptoms of pressure (slowing of the pulse, changes in the disc, vomiting, headache, vertigo, constipation) are easily to be traced back to the hydrocephalus. The fluctuating character of the symptoms is explainable by the changes in the exudate. The phenomena of motor irritation are caused principally by the encephalitis of the cerebral cortex. In semi-pareses and monoplegias one finds frequently, though not always, an especially rich tubercular deposit in the cortical centers concerned, either a *plastic exudate* or an inflammatory softening.

Course of the Disease.—The duration of the disease varies greatly. I have seen patients brought directly from their working place to the hospital die there within a week; on the other hand, I have observed a case in which the patient lay fully six weeks in the terminal stage, and became almost a skeleton, with the chill of death before he died.

As a rule, the actual disease lasts from one and a half to three weeks.

The frequent remissions of symptoms which may appear in any stage of the disease and are only too apt to awaken *false expectations*, are quite striking. The patient may awaken from his stupor, paralyses may disappear, the general condition may even be improved so far that one ventures to think of recovery, only to see all hope destroyed by an early relapse.

While we have seen that the symptomatology is quite variable, there are cases in which the *course of the disease* assumes an entirely *atypical form.* There are, for instance, cases in which *fever* is entirely lacking. I myself have reported such a case in which the temperature did not go above 100 until the very last day. The case started with a hemiplegia alternans superior, so that in the beginning one felt like diagnosing cerebral tumor; actually one of the cerebral peduncles was destroyed through a very deep and extensive tubercular plaque, which manifestly was the cause of the secondary affection of the meninges. Similar cases beginning with monoplegia, hemiplegia, and aphasia have been often described.

It happens also that important symptoms pertaining to the clinical picture of meningitis, as rigidity of the neck, disorders of the cranial nerves of the pupils, paralyses of the extremities, etc., are entirely lacking. I was able to observe this especially in cases which were accompanied by general miliary tuberculosis and ran a rapid course.

Various authors describe cases in which the onset and the course of the disease present the clinical picture of *delirium tremens*. I myself had a case in which the patient was referred to me as being insane. Finally cases are said to be known in which remissions recur and last for years.

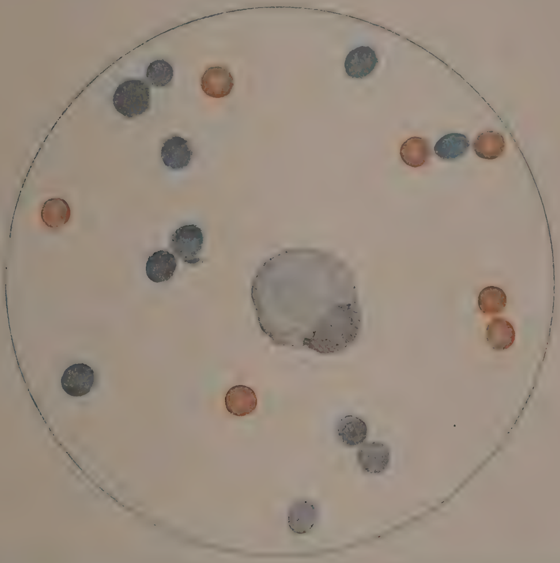

FIG. 162.—Tuberculous meningitis; Nissl's drop method. Moderate leucocytosis, exclusively lymphocytes. Some red blood corpuscles.

On account of the above minute treatment of the symptomatology only little need be added concerning *diagnosis*. The *tubercular* nature of a meningitic disease is proved principally by the existence of a tuberculous process, which, however, in some cases may be found only after a most painstaking investigation, and also by a tuberculous predisposition.

The diagnosis of the *meningitis* can, apart from the symptoms, be verified by *lumbar punctnre*. At a time, when no sure symptoms of a participation of the meninges in the disease can be found, we find occasionally the fluid withdrawn by lumbar puncture changed by inflammation and containing tubercle bacilli. The fluid is usually under a moderately increased pressure; it may be quite clear, but in most cases one sees minute particles scattered diffusely through it. When the fluid settles one can observe a sedimentation of a delicate filaceous coagulum which contains cellular elements as well as tubercle bacilli.

The appearance of the fluid, however, varies considerably; it may appear cloudy, purulent or contain admixtures of blood or other coloring matter; it may be colored an amber yellow (my own observation) or have a greenish tint.

In the sediment, resp. in the coagulum one finds mostly *lymphocytes*, rarely polynuclear leucocytes, sometimes large cells with a vesicular-like nucleus, manifestly endothelia, the presence of which seems to me to have an unfavorable significance. The microscopic findings may vary considerably in different punctures in one and the same case in so far as the number of lymphocytes may increase considerably or that in a later puncture the lymphocytes may be replaced by polynuclear leucocytes. In the case mentioned below in which the patient recovered, the severe pleocytosis disappeared by degrees so that the fluid at the eighth puncture was entirely normal.

The fluid withdrawn by puncture seems *to contain invariably tubercle bacilli*, though sometimes only in extremely small numbers. The work of finding them is, however, very trying and often requires many hours. In tubercular meningitis also one finds not rarely a mixed infection with other bacilli.

The **prognosis is unfavorable.** For a long time the disease was regarded as absolutely fatal. It is true that several decades ago upon pathologic-anatomical grounds (*Virchow*) attention was called to the fact that recovery must sometimes occur, occasional recoveries were also reported by clinicians, but most authors treated these reports skeptically and felt rather inclined to assume a mistake in the diagnosis than to believe in a recovery. Since lumbar puncture, however, has enabled us to make the diagnosis certain, several cases of indubitable tubercular meningitis have been reported which resulted in recovery (*Freyhan*, *Henkel*, *and others*). I myself have demonstrated and described an indubitable case in which the bacilli after lumbar puncture slowly vanished from the spinal fluid which became perfectly normal. This patient recovered a year ago and is doing hard work.

Nevertheless, recoveries must be regarded as rare exceptions and can only be expected in cases in which the primary focus is small and located at a place favorable to recovery.

Treatment.—*Prophylaxis* can be used in all those cases in which removable tuberculous foci exist. In tuberculosis of the testicles, in tuberculous periostitis as well as in tubercular affection of glands, *surgical treatment* is above all to be considered. All the other prophylactic measures which generally are used in tuberculosis are to be applied here. Special care ought to be bestowed on *delicate children* after infection with whooping cough or measles. Swollen glands in children ought to be always regarded seriously and not as a necessary evil. In these cases and especially in cases in which symptoms of the prodromal stage appear, great attention should be paid to nutrition, all the more since the patients in this stage do not care for much food.

If *unmistakable meningitic symptoms* have already appeared, we are usually no longer able to influence the course of the disease. Nevertheless we can improve or alleviate many a troublesome symptom for the benefit of the patient. We can aid physical and mental rest by keeping the room dark and preventing noise during the time of extreme excitability of the sense organs. Thorough clearing of the *bowels* by continual doses of calomel frequently exerts a very beneficial influence on the general state of the patient.

Headache is to be treated with an ice-cap; frequently one must resort to internal medicines, resp. morphine (1/4–1/2 gr.). *Local derivation* by blood letting or application of leeches upon the mastoid processes may bring relief. Inunctions of gray mercurial ointment upon the neck and the vertebral column are much in favor.

The temperature rarely is so high that there is need to treat it medically. As a local antiphlogistic method *Heubner* recommends applying iodoform collodium (15–20%) once daily with a brush on the shaven head after having previously cleaned the cranium with ether.

The same author prescribes iodoform also in a solution of ether (1 : 10) thrice a day ten drops. *Strümpell* advises rubbing the shaven head with a (10%) iodoform ointment.

Some authors praise iodide of potassium (15–45 gr. per day).

When symptoms of irritation are marked besides morphine, hydrate of chloral (in doses of 30–45 gr. in adults, 7–15 gr. in children) in the form of a clyster can be recommended.

None of these measures influence the disease process itself.

The results of *surgical operations*, trephining and puncture of the ventricles (*Ord*, *Waterhouse*), trephining followed by free drainage of the subarachnoid spaces (*Horsley*) do not encourage imitation. Still it is best to have recourse to *lumbar puncture*, which is harmless, every two or three days.

I have frequently observed after lumbar puncture an improvement in the general condition with a lessening of the pressure symptoms. In *Freyhan's* case, after lumbar puncture improvement and recovery followed; in the one case under my observation that recovered, I made eight lumbar punctures and three times *injected into the subarachnoid space* large *doses of tuberculin*. Each injection was followed by marked reaction. Other authors do not regard lumbar puncture as inducing either improvement or recovery. *Continuous washing out of the subarachnoid spaces*, too, has been tried but without success.

D. Meningitis Serosa (Serous Meningitis)

Quite a number of years ago, *Huguenin*, *Oppenheim*, *Eichhorst* and others described individual cases of meningitis with *effusion* into the ventricles of a *non-purulent*, *purely serous* fluid. But *Quincke* while occupied with his lumbar puncture, was the first to find that this form of meningitis is by no means rare; he has described a symptom-complex as an independent disease, designating it *meningitis serosa ventriculorum.* Its principal feature consists in an *abnormally copious serous cerebro-spinal fluid existing under strong pressure, which causes meningitic-like disease phenomena. Quincke* regarded this exudation as analogous to serous effusions into the pleural cavity and denied the parasitic character of the inflammation. Numerous investigators have (following Quincke) studied the clinical picture of this disease, and it has been shown that even if its sole characteristic feature is the serous meningeal exudate, nevertheless it does not possess a uniform character, and has several causes.

Etiology.—The cause of the effusion is, according to *Quincke*, to be found in vasomotor angioneurotic disturbances, as they appear for instance in *migraine or in neurotic edema.*

The *effusion* comes from the plexus chorioideus in *meningitis interna* and from the pia corticalis in *meningitis externa.*

While thus *Quincke* regards the disease in many cases as being primary, others are more inclined to assume that it is rather a secondary affection following infectious diseases. Some authors think that the effusion is caused by *toxic influences. Finkelstein and Pfaundler* in their examinations of infants at the breast could almost always prove the existence of *bacteria* in the cerebro-spinal canal. The *effusion* was mostly observed in the course of cases of *pueumonia and typhoid*, though tuberculosis also seemed to play an important ætiological rôle. Meningitis serosa appears with comparative frequency as a complication of *otitis media* (*Hegener*); in suckling babies it has been observed in the course of *gastroenteritis* and in older infants during *measles or whooping-cough. Traumatism*, as well as *mental overexertion* and *alcoholism* are given as causes of the disease.

As *excitants of the infection*, pneumococci, staphylococci, streptococci, bacteria coli and typhoid bacilli are to be considered. According to *Quincke* the slight virulence and the small number of the microbes in the effusion prevent the formation of pus.

Pathological Anatomy.—In *meningitis serosa interna* one finds a flattening of the cerebral convolutions; the ventricles are widened; the fluid is copious and clear. On the plexus one finds fibrillary layers; the neighboring cerebral substance is œdematous. The pia of the cortex also is usually somewhat inflamed and œdematous.

In *meningitis serosa externa* the latter condition is present to a marked degree; the pia is swollen; microscopic investigation shows an infiltration with round cells, and at the base sometimes also an organized exudate.

The cerebral substance is infiltrated with the serous fluid; in less severe cases there exists a capillary hyperæmia, in more severe ones an anæmia.

The fluid as a rule contains lymphocytes in a relatively small number and sometimes bacteria.

Clinical Picture and Symptomatology.—The clinical picture, as a rule, is neither so severe nor so typical as in the other forms of meningitis. During the *nursing age*, when the disease most frequently appears *secondarily*, especially following disturbances in digestion, the course, it is true, is mostly unfavorable. Thus a form with a "*foudroyant*" course is known, which sets in suddenly with high fever, convulsions and symptoms of *meningitis;* such as rigidity of the neck and pupillary changes, followed rapidly by coma and within a few days or even hours by death.

In *meningitis serosa externa* a larger number of meningitic symptoms can be noted in children than in the interna; the onset of the disease is besides said to be not so brusque.

Ventricular serous meningitis, designated also as *acute hydrocephalus*, is characterized first and foremost by more marked *symptoms of cerebral pressure. Finkelstein* distinguishes two forms; one with an acute onset and the other developing more insidiously and characterized by coma.

In *adults*, too, serous meningitis may appear in different types.

Frequently it simulates but in less severe form the clinical picture of acute purulent meningitis.

The *onset* is sudden, but the fever does not reach a high degree, and abates early; headache and vertigo are less severe. Nuchal rigidity is entirely absent or only slight.

In some cases *fever* is entirely absent or shows a decided irregularity and keeps within moderate limits.

With remarkable frequency *visual disturbances* occur; they may appear in the early stage of the disease and continue after the illness is over. In one case in the Heidelberg clinic, *neuritis optica* was observed half a year after the patient's complete recovery.

Clouding of consciousness is not very marked and may be even entirely absent. In the case I have just cited there was deep coma, from which, however, the patient awoke soon after lumbar puncture was made.

Retardation of the pulse, irregular pulse, Cheyne Stokes *respiration* may be observed also. *Convulsions* are more rare; *Ziehen* saw several cases set in with *Jacksonian epilepsy*.

Spinal symptoms play an unimportant part; abnormal reflexes and variability in them have been observed.

Not rarely serous meningitis simulates a *cerebral tumor*.

Choked disc (reaching even the stage of amaurosis), headache, vertigo, vomiting, even convulsions, paralyses of cranial nerves may make differential diagnosis extraordinarily difficult.

Oppenheim points out two *distinguishing features*, which occasionally help in making diagnosis possible; first the fact that serous meningitis not infrequently has a *congenital foundation* (hydrocephalus) and that therefore the size and the form of the cranium may suggest the right diagnosis, and secondly the protracted course of the disease which with its *remissions* and *intermissions* frequently lasts *for years* and makes a diagnosis of tumor highly improbable.

Paralyses of the basilar nerves are the most important focal symptoms, though they are frequently entirely lacking. In some cases *cerebellar ataxia* is present.

The **course of the disease** is quite varying. Though it is said that in children at the breast serous meningitis usually ends fatally, complete recovery is by no means excluded. The disease may last for weeks and months, and, sparing the life of the patient, lead to *chronic hydrocephalus*. Even this may retrogress later on; but such individuals, as *Quincke* especially has pointed out, are *predisposed* to acute and subacute relapses, which may appear after puberty in consequence of traumatisms, excesses or infectious diseases.

In adults, likewise, after an acute onset of the disease, a transition to *chronic hydrocephalus* may take place. *Oppenheim* observed a case, which extended over nine years. *Quincke* knows of cases of a milder type, in which the patients complain year in and year out merely of headache and vertigo which are usually interpreted as neurasthenia.

Diagnosis is made certain principally by *lumbar puncture*. A liquid as clear as water flows off copiously under strong pressure. It is rich in albumen and shows a tendency to the formation of a web-like coagulum. In the centrifuged fluid, one finds some few uninuclear lymphocytes. The bacteriological examination in some cases may prove the existence of a cause for the inflammation; animal experimentation, too, may eventually be tried to clear the diagnosis. But in such a case it is to be considered that other intra-cranial affections, too, lead to an increase in the quantity and the pressure of the liquor (*Quincke: uræmia, chlorosis, sinus thrombosis*). It is therefore necessary to pay attention also to the history and to judge the clinical symptoms most conscientiously.

The **prognosis** results from what has been said before. Cases of spontaneous recovery are known; but above all, lumbar puncture led to improvement and recovery in many cases. *Oppenheim* thinks that life is the more endangered, the more the clinical picture during the progressive course of the disease approaches that of tumor cerebri. *Quincke* is of the opinion that in those cases, a more favorable course is to be expected in which the albumen contents of the cerebro-spinal liquid does not surpass 0.5%.

Treatment has found the greatest advancement in later years through *lumbar puncture.* It has been tried with success in acute as well as in chronic cases.

There are cases, *Quincke* says, in which the success of *one* lumbar puncture is by far more surprising than that of a pleural puncture, and where the clinical picture is changed at once. This can be observed especially in some acute inflammatory and angioneurotic forms, where manifestly it was only necessary to withdraw the liquid as soon as the exudation had reached its climax.

It must be admitted, however, that more frequently it becomes necessary to *repeat* lumbar puncture at certain intervals.

The quantity to be withdrawn depends upon the degree of the pressure. To withdraw an amount of more than 100 c. cm. at once, I think rather hazardous. It is also necessary to let the fluid drain off slowly, so that a decrease in pressure does not occur too suddenly.

In desperate cases, in which spinal puncture does not produce a sufficient result, one may try also *Neisser's puncture of the ventricles*, which occasionally has shown good results. Also *splitting the dura*, especially in cases of *otitis*, has produced amelioration and even recovery.

As to other treatment, especially in the acute cases, it is identical with that of the other forms of meningitis.

II. LEPTOMENINGITIS CHRONICA SIMPLEX

Etiology.—Simple chronic leptomeningitis is of no great clinical importance, compared with the *syphilitic* form, which will be treated in another chapter.

One may distinguish a *diffuse* and a *circumscribed* form. The former is observed as a part of the symptomatology of a *chronic meningoencephalitis in dementia paralytica* and also in cases of *chronic kidney disease and chronic alcoholism.*

The *circumscribed form* is not rarely the consequence of *traumatism*, which leads to more or less extensive inflammatory processes, followed by indurated thickenings of the pia mater; not rarely in these cases one finds adhesions between pia, dura, and the inner cranium.

Of special significance is the *basilar* form, which not rarely is a consequence of a *serous meningitis.* In many cases the latter is to be regarded as the cause of the interruption of the communication between the ventricles and the subarachnoid space and therefore as the *origin of the chronic hydrocephalus.*

Pathological Anatomy.—There is sometimes a circumscribed, sometimes a more diffuse whitish thickening of the arachnoid and pia mater, which, however, frequently represent but residues of past inflammatory processes. In some cases these changes are to be traced back to continually recurring disturbances of the circulatory and nutritional systems; for instance, in vascular congestions (*Ziegler*).

Circumscribed chronic inflammations characterized by cellular infiltration are usually found in the vicinity of *chronic osteitis*.

When the process is well developed, the pia is cloudy, white, non-transparent, especially in the sulci, along the vessels, frequently also at the top of the convolutions. The affection is principally localized in the fore part of the brain.

Clinical Picture and Symptomatology.—A uniform clinical picture can not be described as very little experience is available relative to this form of meningitis. Most frequently *headache*, *vertigo* and *vomiting occur*. In the *basilar* form, because of a hydrocephalus, one may find out spoken *symptoms of general pressure*. *Paralyses of the basilar nerves* were also observed in some cases, so, too, *Jacksonian epilepsy* where the seat of the induration was at the convexity. With comparative frequency *changes in the fundus of the eye* have been observed.

The headache is sometimes localized at the place of the lesion, it may be also that the cranium at this place is especially tender to pressure. Fever is as a rule absent.

A **diagnosis** can rarely be made with certainty. Confusion with *neurasthenia or traumatic neurosis* is most frequent. The background of the eye may in such cases lead to the right diagnosis. Cerebral tumor also may be thought of. It may be that a preceding traumatism will lead to the right diagnosis. In hæmatoma of the dura mater the symptoms will probably follow the traumatism sooner.

The **prognosis** is mostly favorable quoad vitam, but unfavorable quoad restitutionem, except when the process is so well circumscribed that it can be localized and removed by operation.

Successful *treatment* is only possible through surgical intervention. If *Jacksonian attacks* suggest the seat of the focus, trephining to remove the induration ought to be done.

If the focus can not be localized, nothing is left but a treatment of the individual symptoms, especially of the headache.

3. TUMORS OF THE BRAIN

BY

M. LEWANDOWSKY (Berlin)

In the section on cerebral tumors all those newgrowths are to be treated, which affect the brain; therefore, not only those arising in the substance of the brain itself, but in part also those emanating from the membranes and the skull. Furthermore, in this section are to be treated, not only the real tumors in the pathologico-anatomical sense, but also the granulation tumors, the tubercles and gummata, as well as these other diseases, whose course may exhibit a clinical picture resembling that of tumor, namely the parasites of the brain, the echinococci and cystocerci, also the cysts and one group of aneurisms.

Hæmatoma of the dura mater, which may similarly run its course with symptoms of a tumor, is to be treated in connection with pachymeningitis hemorrhagica and here finds consideration only with respect to differential diagnosis.

Pathological Anatomy

Of the new growths in the real sense of the word, there occur in the brain, most frequently gliomata, sarcomata and endotheliomata.

Neuromata, tumors which consist of the nerve substance itself, of nerve fibres and ganglion cells, do exist, it is true; but they are found mainly, and there, very rarely, in the sympathetic nervous system. In the cavity of the cranium itself, they are very rare and here they never attain any size to make them deserving of consideration.

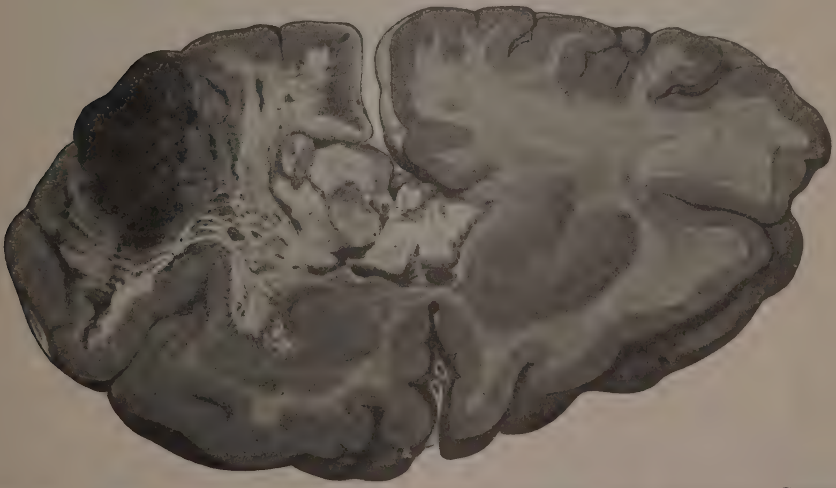

FIG. 163.—Diffuse glioma of the left frontal lobe with hemorrhage into the tumor. General-pathological collection of the Landau clinic. (*L. Pick.*)

Gliomata are the specific tumors of the central nervous system, since they emanate from the gliar tissue which is peculiar to this alone. They consist of glia fibres and glia cells. Occasionally, there are in them spaces surrounded by epithelium, or even rosettes of epithelium, which point to an origin of these tumors upon a foundation of abnormal embryonal processes of development. Gliomata are usually soft and filled with numerous blood-vessels. They belong to the infiltrating tumors and it is almost always characteristic of them, that they spread quite diffusely and without definite boundary into the cerebral tissue (Fig. 163). Microscopically, it may be seen, that in the area of the tumor proliferation, part of the nerve tissue may be more or less preserved. Their consistency is, on the whole, soft, their color, gray, grayish red, or yellowish red, usually irregular; hemorrhages into the tumor are frequent, and through these exiravasations of blood and the consequent changes in the coloring matter of the blood, a many-colored picture may appear in some places. Gliomata may reach almost any size. Occasionally, it is a matter of very extensive diffuse proliferation of the glia, with retention of the exterior contour of the brain; apparently, therefore, the disturbance has led to a hypertrophy of the parts of the brain that are affected and this is not cleared up until a microscopic examination has been made. There are also denser forms of gliomata, which are characterized by a closer weaving of the fibres; they originate most frequently from the ependyma of the ventricle and may then protrude against the ventricle in the form of nodules, but occur more rarely in other places as well (Fig. 164). Especially frequently in the cerebellum, there are peculiar, odd, polypus-like growths, which then compress the adjacent parts of the brain (Fig. 171). Gliomata very commonly undergo cystic degenerations. Metastases occur very rarely, and then appear almost only in the membranes of the brain (Fig. 165). Multiple gliomata are very rare, frequent only in the form which begins in the ependyma of the ventricle.

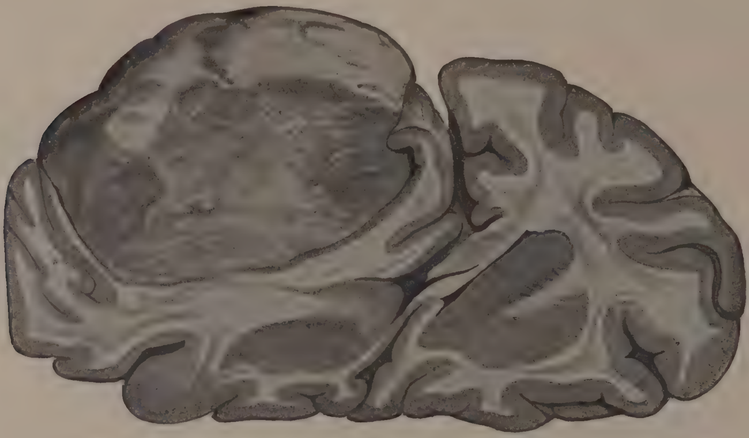

Fig. 164.—Hard, rather circumscribed glioma. General-pathological collection of the Landau clinic. (*L. Pick.*)

With the tumors in the pathologic-histological sense, one must classify also *tuberous sclerosis* of the newly born. The tumor-like proliferations, here, seem to consist of glia tissue and of embryonal nerve cells.

While glioma can originate only in the brain substance, *sarcoma*, which occurs in all its various forms (spindlecelled—roundcelled—smallcelled—giantcelled) usually begins in the meninges, the periosteum or the bone. The growth of sarcoma in the brain is decidedly expansive, and correspondingly it is generally to be distinguished macroscopically from glia by the sharp line between it and the cerebral tissue, which it only pushes ahead of it. Round about the sarcoma, there is frequently found a zone of softening, from which the tumor can easily be dislodged as a whole. The sarcoma may attain a very great size; growing outwards, it can through pressure cause a thinning and actual wasting of the bones of the skull. In the meninges, a diffuse sarcomatosis occurs, which in some cases can be definitely made out only by microscopic examination.

Endothelioma is to be sharply separated from sarcoma histogenetically, even though the differential diagnosis is frequently histologically not quite certain. It originates nearly always in the flat cells which line the inner surface of the dura, or in analogous cells of the soft cerebral membranes, but it may also spring from the endothelium of the blood-vessels or the lymphatics. The cells of endotheliomata frequently have the tendency to group themselves in layers of round or lanceolate formations: in such instances they may also develop a large amount of connective-tissue fibres, and the tumor may then be permeated by calcareous concrements in such a manner, as to make it feel hard, and upon cross-section, as if strewn with sand. It is then called *psammoma*. Psammomata usually do not attain great size (Fig. 166). On the other hand, the epitheliomata which are rich in cells, and are very difficult or impossible to differentiate histologically from the sarcomata (Fig. 167), often attain or even surpass the size of a fist;

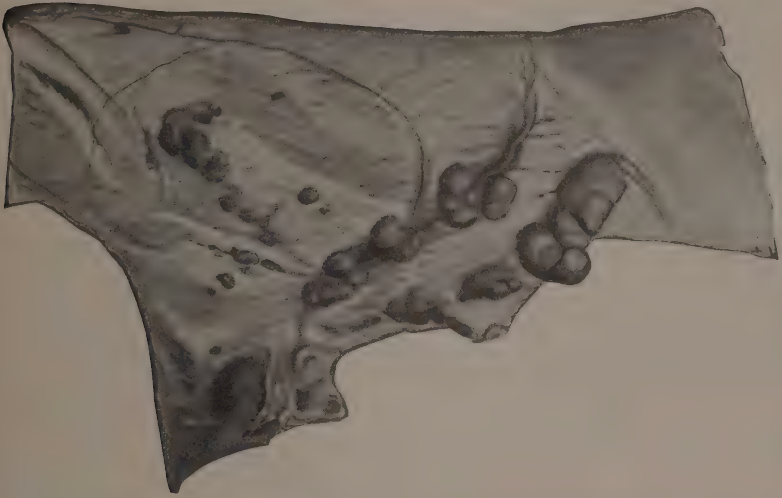

FIG. 165.—Metastatic gliomata of the dura, originating from the tumor in Fig. 164.

as they grow away from the pia, they always push the brain before them, gradually displacing it.

All other tumors may be described as rare.

Of *tumors of the connective tissue*, there occur in the brain, resp. in its membranes, fibromata, lipomata, chondromata, chordomata, angiomata, osteomata, and melanomata (chromatophoromata).

Fibromata are rare, but important as neurofibromata in the cerebral nerves, especially in the intra-cranial course of the auditory nerve. They may be a partial phenomenon of general neurofibromatosis (Recklinghausen's disease).

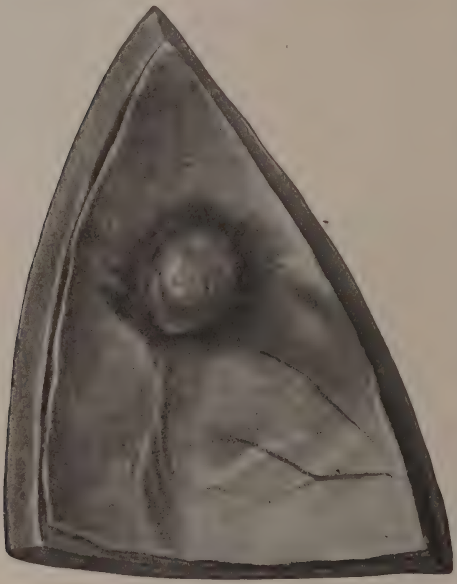

FIG. 166.—Psammoma of the dura. Collection of the pathological institution of the Friedrischshain Hospital. (*L. Pick.*)

Lipomata are very rare, but when they occur they are usually found in the region of the corpus callosum.

Chondromata and osteomata are not more frequent.

The *chordoma*, which arises from the cells of the chorda foundation, occurs exclusively in the clivus Blumenbachii. Here it occupies typically the middle of the bone, and, through a narrow opening in the dura, enters the cranial cavity where it touches the anterior surface of the pons. According to *Ribbert*, it is found in 2% of all cadavers; but inasmuch as it generally does not exceed a pea in size and is of extraordinarily soft, jelly-like consistency, it usually causes no symptoms, except in those extremely rare cases, in which it attains a considerable size.

Melanoma (chromatophoroma) was considered by *Virchow* as a primary tumor of the cerebral membranes, by *Minelli* and *Pick* as a primary tumor of the substance of the central nervous system itself.

The *angioma* is known as teleangiectasis, as cavernoma, and as angioma arteriale racemosum, but is rare in all forms.

Besides the tumors of the connective tissues, there are found, but less frequently, *epithelial tumors; adenomata* and *carcinomata* may develop from the epithelium of the ventricles. Adenoma of the hyphophysis, the usual form of tumor in this place, is the only one more frequent (Fig. 173).

Furthermore, the *dermoid cysts*, *the cholesteatomata*, and the *teratomata* (Fig. 170) which may all be traced to embryonic germinal displacement, are not very uncommon. The cholesteatomata owe their origin to the embryonic misplacement of epidermis in the pia (*Bostroem*). They are usually found at the base (Fig. 168) or in the ventricles, and show macroscopically accumulations of the well-known scales which shine like mother-of-pearl.

Metastatic tumors in the brain, especially carcinomata, are not very rare. The cerebral membranes are even more frequently attacked than the brain itself.

The *granulomata*, gummata and tubercular swellings, are also very frequent forms. The *gumma*, in the majority of cases, emanates from the meninges. It appears upon cross-section sharply defined against the brain substance, which it pushes away, rather than infiltrates. Its favorite seat is the convexity of the cerebrum. In rare cases, it may reach the size of an apple, but is usually smaller. It shows on the one hand, a tendency to caseation, on the other, to the formation of indurated scars. We must also mention gummatous meningitis of the base, which may run its course under the picture of basal tumor formation.

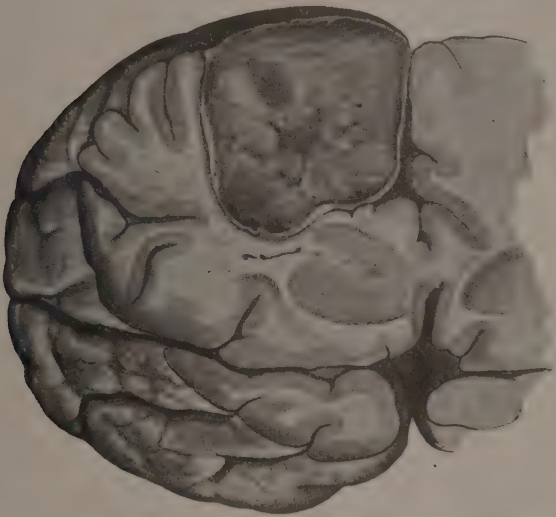

FIG. 167.—Endothelioma, originating from the pia. Collection of the pathological institution of the Friedrichshain hospital. (*L. Pick.*)

The *solitary tubercle* may resemble the gumma macroscopically, because it shares with it the tendency to caseation. Unlike the other, it usually appears in childhood and prefers the brain stem and the cerebellum, regions in which the occurrence of gumma is rare. The tubercular swelling, furthermore, shows an inclination to caseous purulent softening, and may then create the impression of an abscess, the nature of which can only be cleared up by bacteriological examination.

Of the granulation tumors caused by actinomyces (*actinomycoma*) very few cases have been described (*Bollinger*).

Tumor formations caused by *yeast organism* were described first by *Krönig-Hansemann*, and since then, in a few other cases.

Of the *parasites* of the brain, the *cysticercus*, the scolex of tænia solium has become rarer and rarer since the introduction of meat inspection. In the sixties of the preceding century, about 2% of the bodies, on which postmortem examinations were made in the Berlin Charité, were infected with cysticerci; in 1900 this percentage had fallen to 0.15%. The brain is by far the most common seat of the cysticercus; it generally locates itself in the cortex, the membranes and the ventricles. It is possible that only one cyst is found, as is particularly often the case with cysticercus of the fourth ventricle, but usually there are several, or many (Fig. 169). The single cyst, at the end of its development, appears as a small vesicle filled with a fluid as clear as water, its size varying from that of a cherry pit to that of a hazel nut, at the outer end of which there is a contraction, the sign of the location of the future tænia head. This simple vesicle, however, in the ventricles (Fig. 173) and still more in the arachnoidal space of the base, may assume the most extraordinary forms, in that it may attain an

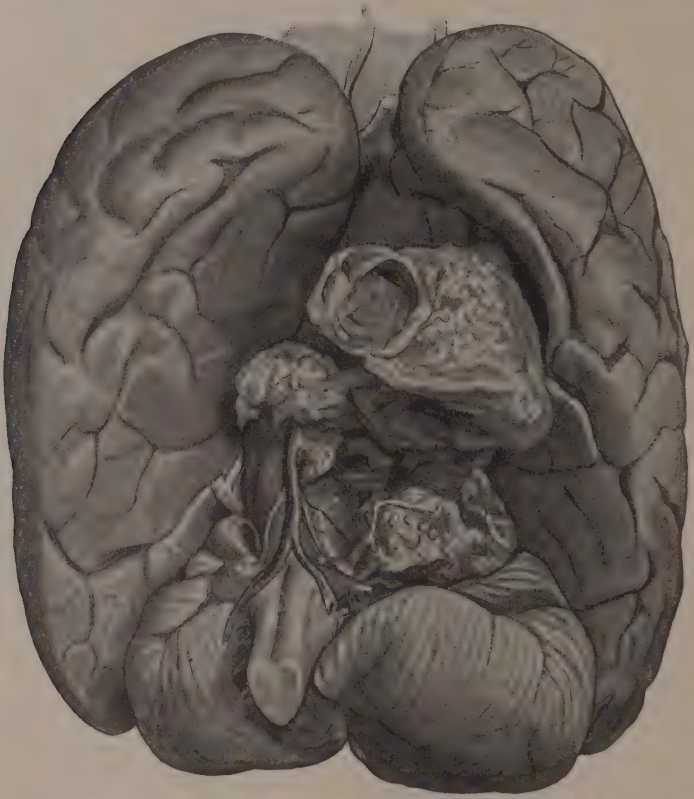

FIG. 168.—Cholesteatoma of the base. General-pathological collection of the Landau clinic. (*L. Pick.*)

abnormal size through manifold outspreading subdivisions and branches. Especially at the base, the cysticercus vesicle is inclined to progress beyond the vessels into the crevices of the base and the fissures of the brain. The nature of this extraordinary formation was first explained by *Zenker* as cysticercus racemosus through finding the head of the scolex. As a result of the irritation produced by the growth of the vesicle, there may occur inflammatory proliferation on the part of the soft cerebral membranes and even the formation of fibrous masses which may now completely cover the collapsed cysts has been observed, so that the exterior picture of a fibrous or gummatous basal meningitis may arise (basal cysticercal meningitis). Older cysts not only such as have been closed, but also those which have branched out, may become calcareous, but, on the other hand, cysts may live and maintain themselves in the body for decades.

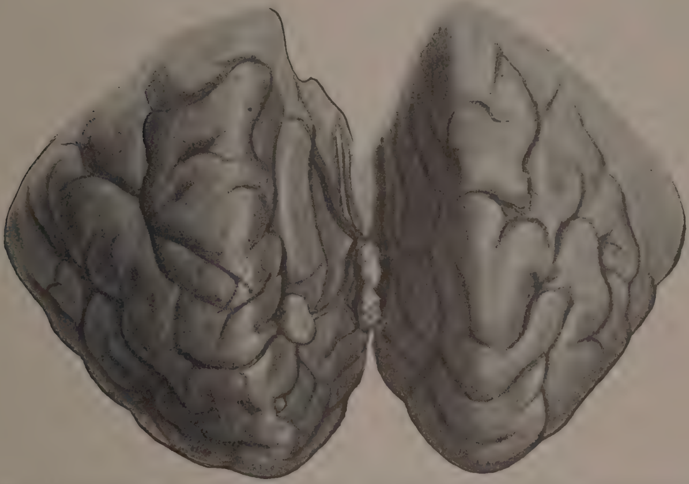

FIG. 169.—Multiple cysticerci. Preparation of the general-pathological collection of the Landau clinic. (*L. Pick.*)

Echinococci in the brain are much more uncommon in Germany than the cysticerci. Several cases are known, in which the echinococcus cyst caused a thinning in the cranial bones and broke through the surface (autotrephining).

The non-parasitic *cysts of the brain* (with the exception of the dermoid cysts) owe their origin to more serious disturbances of the brain, i. e., encephalitis or traumatism.

Of the aneurisms only the larger can give rise to tumor-like pictures. They occur most frequently in the arteries of the base of the brain, especially in the a. fossæ Sylvii; they occur also in the internal carotid itself, in the basilar and vertebral arteries, etc. They may attain or even exceed the size of a hen's egg.

The development of larger tumors often leads to very considerable dislodgment and displacement of the cerebral parts (Figs. 164, 165), as well as frequently, in consequence of the increased pressure upon the brain, to hydrocephalus (cf. below symptomatology).

Occurrence and Etiology

Tumors of the brain seem to occur much more frequently in males than in females. Several authors assert that about two-thirds of all cases are observed in males. Concerning the age of the persons affected it can only be said that the frequency of cerebral tumors decreases considerably after the 40th year. Nevertheless isolated cases occur even at the most advanced age. Cerebral neoplasms are particularly frequent in children, especially if tuberculous growths are included.

We know about as much and as little of the *ætiology* of brain tumors, as we do of that of tumors in general. It is quite superfluous therefore to enter here upon any theory as to their origin. Just as with all other tumors, however, we are nearly always under the impression with those of the brain, that they are the result and development of an *abnormal predisposition*, and that even if they do not develop until an advanced age, they have been laid by an inexorable fate as a gift into the cradle of the sufferers. Heredity plays no part at all in their development. But their formation from an abnormal predisposition is really quite clear in the choleastomata and related tumors and in the gliomata also we have already pointed out certain indications, that make a similar hypothesis probable. But even if we can not assign a real cause in the majority of cases, the question recently has gained much in practical interest, as to how much an *accident*, especially a traumatism of the skull, may have to do with the development of a cerebral tumor.

It seems as if even in a scientific sense, such connection between traumatism and the formation of a cerebral tumor is not to be rejected off hand, since a connection between a traumatism and the development of a tumor is recognized in the tumors of other organs also. Of course the presumptive evidence for the scientific proof of the traumatic origin of a cerebral tumor is to be found only in a very few cases. First of all, there must have been perfect freedom from symptoms before the accident. In most of the cases it seems in all probability, that the traumatism was rather a consequence than the cause of the tumor; say, in a fall due to an attack of vertigo. It is also of considerable importance that the time intervening between the traumatism and the development of the tumor is not excessively long. In those cases, in which the tumor appears on the site of the cranial trauma, a connection is particularly probable.

But for practical determination, in fixing the amount of damages due the plaintiff, it is impossible to apply this strictly scientific standard. The man injured by an accident, is naturally interested in concealing symptoms of disease that were present before the accident and, if such can not be established definitely, it is impossible to make any other decision, but that—since, in theory, the origin of a tumor on the basis of an accident is possible, such a connection seems not to be excluded in the case concerned. After all, even if symptoms of disease are shown to have existed before the accident, the question still remains, whether the symptoms of the tumor have not been considerably aggravated by the accident. We find ourselves, therefore, in practical determination of the traumatic origin of the tumor exactly in the same position we occupy when considering a large number of other diseases of the nervous system.

The cause of the disease is naturally evident in the cases of tubercular and syphilitic granulation tumors. The determining cause for the development of these tumors in the brain, or, in other cases, the reason for their considerable aggravation may again be a traumatism, especially a cranial trauma. *Cerebral tubercle* may seem clinically to be the first manifestation of tuberculosis; but they probably always arise from tubercular disease of other organs, especially of the lungs and the lymphatic glands. *Gummata*, as is well known, can arise even decades after the syphilitic infection, apparently during a condition of perfect health, without the possibility of demonstrating clinically any other sign of present syphilis. Also upon a foundation of hereditary syphilis, cerebral gummata may occur in childhood or even up to the time of puberty.

The mechanism of cysticercus and echinococcus infection, we may here assume to be well known.

General Pathology and Symptomatology

The tumor itself is a locally restricted disease of the brain, but in the great majority of cases it leads to consequences, which, independent of its special location, are far more dependent upon its influence on the entire brain and its cavities. Therefore, we distinguish *local symptoms* from the *general symptoms* and wish to mention here, that by far the majority of the general symptoms are the result of the augmented pressure upon the brain, the greater tension and the increase in the cerebro-spinal fluid. The so-called "neighborhood" symptoms (Nachbarschaftssymptome: accessory, contiguous, symptoms) arise from the fact that the tumor, differing absolutely from, let us say, a hemorrhage, the longer it exists, the more it affects, though in each case in a very different degree, its vicinity and injures it by the pressure it causes. The compression which is caused by the tumor, has an especially injurious effect, when it affects structures, which can no longer yield, which, as a matter of fact, are directly adjacent to the osseous cranium. These, first and foremost, are the cerebral nerves at the base, which therefore play an especially important rôle in the study of the "neighborhood" symptoms.

The **course of the disease** in the genuine cerebral tumors, especially in the gliomata and sarcomata, progresses slowly towards death. In such cases the general symptoms very frequently overshadow the local symptoms. The most frequent incipient symptom is headache, frequently associated with complaints similar to those of the neurasthenic. General epileptic convulsions, also, which need differ in no respect from genuine epilepsy, may precede the local symptoms for years, but still are a rather infrequent incipient symptom. Of the ordinary symptoms is *increased brain pressure* with all its concomitants to be discussed below, but especially the visible expression of the cerebral pressure, choked disc, frequently a very early sign, especially in the expansive, growing tumors in the posterior cranial fossa. But absolute laws are not to be found here, in any direction; we shall call attention later on, to certain rules. In a smaller group of cases, the local symptoms step into the foreground from the first and there are naturally all combinations of local and general symptoms. The objectively demonstrable local symptoms predominate naturally, all the more, the more important is the area affected by the new formation, or—in the region of the cerebrum especially—the more supplied with projection fibres this area is. Not infrequently irritation symptoms, in tumors of the motor region epileptic fits of *Jacksonian* type, appear even in the beginning of the development and no other disease of the brain leads so frequently to such irritation phenomena as cerebral tumor. A regular progression in the course of the disease is the exception. Exacerbations, especially those of the general symptoms, lasting a variable length of time, may appear; they disappear again in part, but frequently leave behind them permanent deterioration. Complete intermissions, which simulate recovery, occur only rarely. Of the local symptoms, the phenomena of functional loss frequently advance continuously, whereas the irritation phenomena, for instance the *Jacksonian* convulsions, appear only at intervals, but may occasionally appear in groups. Very acute aggravations are sometimes the result of a hemorrhage in the tumor, or even of a sudden increase in the cerebral pressure. The size that a tumor must attain, to produce phenomena, varies considerably, and depends mostly on its location. Tumors as small as cherry pits, in the motor region, may cause convulsive attacks, and those not much larger, in the neighborhood of the fourth ventricle may even bring on death. But, on the other hand, tumors have been found as large as apples, especially in the less important parts of the cerebrum, but also, even though rarely, in the posterior cranial fossa, which caused no symptoms, or, at least, in no way disturbed the patient's ability to work or his well being. In such cases, the disturbances may sometimes begin rather suddenly and advance rapidly. In rare cases even still larger tumors are a secondary finding in the postmortem, naturally, only when they are located in the "silent" parts of the cerebrum. Apart from the location of the neoplasm, the rapidity of the course depends on the rapidity of its growth, which is greatest in the soft sarcomata and in certain gliomata; other forms of gliomata grow more slowly. The fibromata and some endotheliomata (psammomata) grow slowly; they may even come to a standstill in growth, like the angiomata and enchondromata, so that the symptoms come to be stationary. The cysts, too, naturally may be stationary, in some cases causing symptoms only from time to time. The course of tubercular swellings is, on the whole, rapid, but through calcification, they may even recover clinically. Gummata especially may recover, seldom spontaneously; more frequently through internal medication. Finally, an interruption in the course may be brought about by an operation. The duration of the course in the most commonly occurring gliomata, sarcomata, and endotheliomata, amounts, on an average, to from six months to two years. But tumors have been observed that persisted for decades.

The parasitic diseases, i. e., echinococci and cysticerci may have a course exactly similar to that of genuine tumors. They remain stationary, however, more frequently. Cysticerci may, as has been already observed, undergo calcification and are then, unless located in an especially important place, an accidental postmortem finding, after having caused in life sometimes only transient symptoms, at other times no symptoms at all.

The aneurisms also may run a course like that of a genuine tumor and gradually lead to death. In many cases, death supervenes through rupture. In other cases, recovery occurs through organization of the aneurism, though occasionally, only after the aneurism, by its growth, has led to permanent cerebral disturbances.

In by far the majority of cases, death is the *final outcome* of cerebral tumor. It occurs usually in a condition of deep coma, after all stages of dimming of consciousness have been passed. The centers of the medulla oblongata, which are essential to life, gradually fail to perform their task, particularly under the influence of increased brain pressure; respiration becomes insufficient, may finally present the *Cheyne-Stokes* type, the vagus centre becomes paralyzed, swallowing is made difficult, partly because of direct injury to its bulbar centres, partly because of the clouded intellect, which causes faulty swallowing. Thus, sub finem, pneumonia may develop, and the exhaustion resulting from the violent pains, as well as the impossibility of sufficient nourishment, because of the vomiting and disturbances in swallowing, may hasten death.

In a minority of cases, death occurs *suddenly*, especially frequently, but not exclusively so, in tumors of the posterior cranial fossa, which may suddenly paralyze, by pressure, the respiratory centre. The same thing, however, can be effected by a sudden increase of the general cerebral pressure without local affection of the posterior cranial fossa, as, e. g., in tumors of the frontal brain. In rare cases, severe hemorrhage from the tumor, especially if it breaks through into the ventricle, may cause sudden death. A solitary tubercle frequently leads to tubercular meningitis through infection of the cerebral membranes and thereby to death.

After this description of the general course, it is to the point to discuss, now, *the pathology of the general symptoms.*

By far the greater number of the general symptoms are the result of the *increased brain pressure*, i. e., of the increased tension of the cerebro-spinal fluid, which is also nearly always accompanied by an increase in that fluid, and which leads to the development of a hydrocephalus internus. Upon autopsy, then, we see, besides the dilatation of the ventricles, flattening of the convolutions of the brain, the blotting out of the sulci, congestion of the veins of the pia, and pallor of the substance of the brain. When the brain is laid open freely, for purposes of operation, the same observations may be made, and even before then one notices in most cases, that the markedly drawn and tense dura does not pulsate. At the autopsy the cerebral nerves usually seem flattened by the pressure. In children, the brain pressure may result in a bursting apart of the bones of the cranium, which have not yet firmly united. In some cases, it has been observed that the fluid has made a way for itself through the ethmoid bone, thus bringing about its continuous escape through the nose.

How a tumor, which very often develops in a small area of the brain, may give rise to a general increase in the tension of the cerebro-spinal fluid, has not yet been wholly explained. Spacial restriction alone, is, however, not the cause; for, experimentally, we can insert large masses of wax, or a similar substance below the dura, without giving rise to a general increase in brain pressure. The result of this procedure is only, that the brain is locally compressed and injured. One must therefore suppose, that for the production of increased brain pressure, there is needed the constantly acting expansile tendency of the growing tumor, from which the pressure is transmitted throughout the brain. The increase in tension of the fluid would then be the result either of increased production, or, probably, still more of an impaired absorption of the cerebro-spinal fluid.

It is an old moot point as to how one must suppose the increased pressure of the cerebro-spinal fluid (or even the direct pressure influence of the tumor) affects the cerebral tissue. *v. Bergmann* advocated the view, that the brain pressure could directly influence the functions of the nervous system through the production of capillary anæmia, since the brain may be regarded as incompressible within the rigid cranial walls, and pressure could exert an influence only by crowding out the vessels. On the other hand, *Albert* and others insisted that it is of no consequence, whether the brain is or is not compressible in a physical sense; it suffices that if under the influence of pressure the parts mutually crowd one another out, the convolutions may become flattened, just as we see it postmortem in patients with tumors and hydrocephalus, so that one must assume that the connection of the elementary parts is severely impaired. Therefore we must probably assume, that the direct injury of the brain substance also plays a rôle in the origin of the so-called brain-pressure symptoms, besides the difficulty in circulation, which undoubtedly likewise has an effect. This latter, as *Cushing* demonstrated from direct observation of the brain through a glass window, which he inserted in the cranium, consists first, in a compression of the veins, in which the slightest degree of pressure is present, and which therefore indicates congestion. If the increase of the brain pressure still continues, the blood is pressed out of the capillaries, and anæmia results. As *Kocher and Cushing* emphasize, at a certain stage of brain pressure there now results from this anæmia, a stimulation of the vaso-motor centre in the medulla oblongata, which responds with increase of arterial pressure, which in turn overcomes the capillary anæmia. But the result of that is, then, that the stimulation of the vaso-motor centre ceases again, the blood pressure falls again, the vessels of the brain again become anæmic; this action may continue very long, in fact so long that the cerebro-spinal pressure affects the vessels so powerfully, that the vaso-motor centre can do nothing to oppose it. The arterial blood pressure, therefore, works against the brain pressure, and as *Naunyn* proved experimentally, it corresponds to this fact that a lowering of the pressure of the blood accelerates the symptoms of brain pressure.

Hydrocephalus may be particularly pronounced in tumors of the cerebellum. As cause, it has been assumed that the compression of the vena magna Galeni, which conducts the blood from the plexus choriodei into the sinus rectus, creates venous congestion and especially poor conditions for the absorption of the fluid. Of the tumors that develop in the substance of the pons, it is known, on the contrary, that they nearly always give rise to brain pressure either very late, or not at all.

Since the introduction of lumbar puncture by *Quincke*, we have a means of directly measuring brain pressure. While the pressure, measured in a supine position, amounts normally to about 120 mm. of water, it may, in brain pressure reach 1000 mm. water and more. Pressures up to 200 are probably still within the limits of the normal. Pressures from 200 to 250 are very suspicious, and those above, certainly pathological. Naturally it need not be mentioned, that the determination of cerebral pressure does not prove by any means the presence of cerebral tumor. Brain pressure may also be ascertained by puncture of a ventricle.

The obstruction of the aquæductus Sylvii by tumors located in its vicinity, naturally hinders or prevents communication between the ventricles; hence it may happen that lumbar puncture either from the beginning, or after the removal of small quantities of fluid shows rapid decrease in pressure, while ventricular puncture may show considerable pressure and large quantities of fluid.

The immediate expression of the brain pressure is *choked disc*, of the importance of which we owe our knowledge to *Graefe*. We consider it certain, that in the sense of the *Schmidt-Manz* theory, the mechanical influence of the brain pressure suffices for the production of choked disc.

Since the sheath of the optic nerves is continuous with the arachnoidea, the increase in brain pressure causes the cerebro-spinal fluid to be pressed against the lamina cribrosa; this becomes edematous and bulges out, whereby may arise compression and congestion in the vena centralis retinæ.

The occurrence of unilateral choked disc especially speaks for the mechanical theory, as it can be explained only by the local influence of the pressure on the lymphatic vessels of the one eye. That, on the other hand, a neuritis optica may be caused by purely chemical influences, we are taught by its occurrence in polyneuritis and a series of other diseases. But in tumor, this origin from a chemical cause is probably not to be considered.

But since, in any case, neuritis optica need not always be the expression of brain pressure, the condition of the measurable brain pressure beside the neuritis optica is always of special importance. If the neuritis optica is the result of an increase in brain pressure, we shall be able to ascertain this in all cases, by lumbar puncture as well. Occasionally, the increase in brain pressure clearly precedes the choked disc. Pronounced papillitis without brain pressure must indicate that some disease other than cerebral tumor is present. Only if the tumor should be so located that it itself exerts the pressure upon the lymphatics of the opticus, could such a case occur with tumor present; but, as a matter of fact, such a case has not yet been actually reported.

Choked disc, relative to itself alone, may disappear so completely that after a few months scarcely anything abnormal can be found upon ophthalmoscopical examination. But this improvement occurs only in those rare cases, in which the original disease has been cured, or there is, at least, disappearance of the brain pressure. Otherwise, there develops sooner or later, sometimes with considerable rapidity, a neuritic atrophy from which there is no longer a recovery.

Visual disturbances may long be lacking, though choked disc is present. On the other hand, when atrophy appears, more severe disturbances have been observed, which frequently progress to total blindness.

The question has been asked whether processes, analogous to choked disc occur, through cerebral pressure, *in the other cerebral nerves* as well; and this has been answered affirmatively for the auditory nerve (choked labyrinth). But so much only is certain, that injuries of the cerebral nerves may arise from brain pressure, which may cause diminution or loss of their function: it is, however, doubtful whether the mechanism is the same as that of choking in the optic nerve. It is much more probable, that it is a matter of the results of simple compression. That brain pressure may have an injurious effect upon *the roots of the spinal cord* also, is important to note. The most frequent of the disturbances thus caused is the loss or the diminution of the tendon reflexes through injury to the posterior roots, which is manifested in a more or less considerable degeneration of these, resp. the posterior columns. But besides the loss of the tendon reflexes, other serious disturbances, as, for instance, ataxia, do not generally occur. Motor paralyses, also, for instance in the peroneus region, are occasionally observed as the result of an injury to the anterior roots.

The expression of the general disturbance in the functions of the cortex of the brain, which is caused by the brain pressure, is the *stupor* which is the characteristic psychic factor in the general condition of the tumor patients. From a slight feeling of difficulty in recollection and of dullness, which is at first more subjectively present to the consciousness of the patient than would appear, especially from a single examination—from these slight disturbances then, to the deepest sopor, in which all conscious reactions are lost, and feces and urine are evacuated involuntarily, all degrees are to be found. An overwhelming desire for sleep is very usual, even in the beginning of the disease. It is possible, but has not been definitely proved, that in cerebral tumor, besides the stuper other forms of psychic disturbance, to be mentioned later, are caused by the brain pressure. The more severe degrees of brain pressure always cause stupor.

More or less sudden attacks of unconsciousness, which may be ushered in by symptoms of vertigo, are not altogether infrequent. Under the word "vertigo" are comprised, it is true, an entire series of by no means homogeneous, subjective symptoms: darkness before the eyes, rudimentary fainting fits, slight conditions of stupefaction, uncertainty on the feet—all this is described by the patient as vertigo. Genuine rotary vertigo, is rare, as a general symptom.

Headache, almost uniformly a symptom of cerebral tumor, is very probably caused by the brain pressure, not upon the substance, but upon the membranes of the brain, and especially upon the dura, which is stretched and pulled by the brain pressure, and which, as is well known, is extraordinarily sensitive to influences of all sorts. The headache in cerebral tumors is in its more severe forms dull, boring, overwhelmingly violent, can not be exactly located, even though it is said to be more marked on one side, and frequently, too, in the forehead and the back of the head. Sudden increase in the headache is usually accompanied by an increase in stupefaction. That the headache in the majority of cases is dependent on the brain pressure, is proven by the fact, that by lessening the pressure by means of lumbar puncture, we can, for a time, ease it considerably in most cases, or even remove it. It is, of course, self-evident, (and this we shall discuss later on), that a tumor by direct irritation or stretching of the dura, in a circumscribed spot, may also cause headache, independent of the general brain pressure.

Besides headache and stupor, *retardation of pulse*, and *vomiting* are among the leading classical general symptoms in cerebral tumor. They are general symptoms inasmuch as they, too, are dependent on brain pressure; but in contra-distinction to the stupor, they are dependent upon the *local* mechanical irritation, which the brain pressure exerts on a circumscribed place, especially on the structures in the floor of the fourth ventricle. Here is stimulated the vagus center of the heart, which retards the heart beat, and the vomiting center, which especially through the vagus, but also through the splanchnic, influences the stomach; furthermore, as is well known, it arouses the abdominal pressure into activity, in the way characteristic for vomiting. The vomiting of the tumor patient is almost independent of ingestion; if there is food in the stomach, naturally, it is vomited; otherwise, what is cast up consists only of mucus mixed with bile. The tongue need not be coated, and the appetite may be fairly well preserved.

Hiccough and frequent *yawning* are rarer general symptoms of cerebral tumor, which, like those named before, are the result of irritation of the centers located in the medulla oblongata. *Respiratory disturbances* have already been mentioned as final symptoms, but may occur, from a similar cause, as transitory symptoms.

Diabetes mellitus and diabetes insipidus, also, can occasionally occur, even without local pressure of a tumor on the centers concerned on the floor of the fourth ventricle, simply by the effect of the brain pressure upon them.

The genesis of the symptoms discussed thus far and resulting from the increased brain pressure, is easily explained. They are due to the influence of this pressure either on the entire brain and its membranes, or upon such individual parts of the brain, which, as a result of their location, and possibly of a peculiarly sensitive predisposition are especially exposed to the influence of this general brain pressure; this is particularly true of the centers of the medulla oblongata.

But there are still other injuries due to the general brain pressure, which are difficult to explain, because they occur only in a portion of the cases, and because they differ in each individual instance. For it may be regarded as certain, that the brain pressure may, upon occasion, produce almost any local symptom, though even post mortem, no cause can be discovered for this local injury, which evidently must have existed. It is therefore proper to speak of *localized symptoms of general brain pressure*, even though we can not say, why brain pressure in one case should cause paralysis of a cerebral nerve, in another *Jacksonian* epilepsy, and in a third no localizing symptom

Stitt. Tropical Diseases

Synopsis of the Contents. Diseases due to protozoa; Malaria, Blackwater fever, The trypanosomiases, African trypanosomiasis, Brazilian trypanosomiasis, Tropical relapsing fevers, Leishmaniases, Visceral leishmaniasis, Cutaneous leishmaniasis. General considerations of dysentery, Amœbic dysentery, Liver abscess. Diseases due to bacteria: Bacillary dysentery, Plague, Cholera, Malta fever, Leprosy. Diseases due to filterable viruses: Yellow fever, Dengue, Dengue like fevers, Phlebotomus or Pappataci fever, Seven day fever, Sand fly fever and Three day fever, Six day fever. Food deficiency diseases: Beriberi, Rice and Beriberi, Pellagra, Sprue, Hill diarrhœa. Helminthic Infections: Ancylostomiasis, Filarial infections, Filaria Bancrofti, Filaria loa, Filaria volvulus, Filaria medinensis, The schistosomiasis, Japanese schistosomiasis, Minor helminthic infections, Paragonomiasis, Clonorchiosis, Intestinal distomiasis, Cochin-China diarrhœa. Infectious Granulomata of the Tropics: Yaws, Gangosa, Mycetoma, Granuloma venereum. Tropical skin diseases: Tropical ulcer, Tinea imbricata, Tinea curis, Pinta, Minor tropical affections of the skin, Dermatophiliasis, Tropical impetigo, Piedra, Cutaneous myiasis, Creeping eruption, Craw-craw. Tropical diseases of disputed nature or minor importance: Verruga Peruviana and Oroya fever; Heat-stroke and Heat prostration, Tsutsugamushi or Japanese River fever, Spotted fever of the Rocky Mountains and tabardillo or Mexican typhus, Climatic bubo, ainhum, Goundou, Rat bite Disease. Diagnostics: Onset and the temperature chart in diagnosis, Blood examinations in diagnosis, Circulatory, respiratory and lymphatic systems together with anæmia, hæmorrhages and œdema, Jaundice and the liver and spleen, Urine and the genito-urinary apparatus in diagnosis, The feces and alimentary tract, Neurological considerations.

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DIAGNOSTICS AND TREATMENT

of Tropical Diseases

By E. R. STITT, A. B., Ph. G., M. D.

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at all. That these symptoms need not depend upon indefinite distant effects of a tumor is proven definitely by those cases, in which an increased brain pressure was caused by disturbances other than tumor. Only in a minority of cases, is a localization of the brain pressure, resp. the hydrocephalus itself on one side of the brain or of a ventrical—or a local meningitis a sufficient explanation for these localized symptoms of the general brain pressure. They are most likely the result of the configuration of the cranium, of the greater or less pliability of the protective envelopes, or they must depend upon individual differences in the supply of blood vessels. In the majority of cases, these localized symptoms of the general brain pressure consist in a paresis or paralysis of one of the basal cerebral nerves, for instance, the abducens or the oculomotor, etc.; usually these disturbances are unilateral. Furthermore *Jacksonian* convulsions occur, because of local irritation of a motor zone due to brain pressure; also hemiplegic disturbances, aphasia, and very frequently cerebellar disturbances. Now if such local disturbances are caused by brain pressure in cases in which no tumor at all exists, they naturally may be caused in the same way when a tumor is present; thus local symptoms are present, if not very commonly, still not infrequently and in practically important cases, which have no connection with the tumor, but are only localized disturbances of the brain pressure caused by the tumor.

Finally, it is important to know that cerebral pressure, in most cases, is not of a constant, nor evenly increasing degree, but that at some times—for hours or days—it may present considerable *increase*, which can also be demonstrated objectively by lumbar puncture. These increases are made manifest by the augmentation of the symptoms dependent upon the brain pressure; the headache increases, the stupor deepens, the retardation of the pulse, which on the whole is very rare as a permanent disturbance, becomes more pronounced, etc. At the height of these variations in brain pressure, there then appear frequently the localized consequences, which we have but now discussed, and which may remain then either permanently, or may again disappear. Thus, there is a transient amaurosis, which is explainable by the compression, which the chiasma and the tractus optici suffer by pressure from the third ventricle.

So much relative to the symptoms, which are dependent upon the increase in brain pressure.

Less important are those symptoms which are dependent upon mechanical irritation, which a tumor naturally causes in the contents of the cranium and which is difficult to define, or which possibly can be explained by chemical metabolic products of the tumor. We include among these, the *general epileptic paroxysms*, which, as has already been mentioned, may precede by a long time all the other tumor symptoms. Perhaps they occur with particular frequency in multiple tumors; thus, multiple cysticerci may exist for decades under the picture of an epilepsy without any of the symptoms of increased brain pressure.

A whole series of *psychic disturbances*, which are observed in cerebral tumors, such as sudden and furious attacks of anger, melancholy moods, conditions of light hypomania, very rarely, hallucinations, are not necessarily to be explained by the agency of a general brain pressure and can not always be thus explained. Temporary delirious conditions are frequent; the development of the *Korsakow* symptom complex is rare. We shall discuss later, in speaking of tumors of the frontal lobes, whether a psychic disease may be a local symptom of a tumor. Finally, hysteric symptoms may supervene upon the tumor symptoms proper.

As to the *general character of the local symptoms of tumor*, they correspond naturally, on the whole, to the facts disclosed by investigations into brain localization. To these investigations, observations of the course of cerebral tumors have contributed considerably. But, in general, the local symptoms of brain tumor are far more difficult to judge than those of other local cerebral diseases. For though hemorrhage and softening do not always exert their influence only in the place of their origin, but also in the vicinity of the area directly destroyed by them; though they may even temporarily lead to serious general phenomena, still the fact can always be depended upon that those focal symptoms that occupy the central point in the clinical picture correspond also to the main seat of the disease, and that those cerebral areas, in which we must localize the functions that have been retained, are also intact anatomically. The development, the manner of the growth of the tumors, as well as the combination with the general symptoms described above, so arrange matters that we do not possess the same certainty in tumor and that we may be deceived in two opposite directions. On the one hand, it occurs, that not only small, but even very large tumors cause no or only very slight symptoms; either because they only push the brain aside, or if it concerns infiltrating forms, because in their area some of the nervous substance still remains functionally capable. It is extraordinarily surprising to see what deformations and displacements even of vital parts of the brain, for instance of the medulla oblongata, are sometimes caused by a tumor, without the appearance, of serious disturbances in the functions of these parts. This is especially the case, when very slowly growing tumors, possibly those existing since early youth, are concerned, and the brain has thus gradually been able to accommodate itself to the abnormal conditions. Precisely for this reason, therefore, negative findings in tumor frequently prove nothing for the physiological localization of the cerebral functions.

The other peculiarity of the tumor, that its effect frequently extends far beyond its own location, works in the opposite direction. Not only is a zone of softening found around certain tumors, but the tumor, by the pressure it exerts, injures in a larger or smaller area, the functions of cerebral parts in the neighborhood. These "neighborhood" symptoms are probably in general under otherwise equal conditions the more pronounced, the greater the general cerebral pressure is, and to the damaging effects of which, the effects caused locally by the tumor itself have to be added. The "neighborhood" symptoms may extend either to the parts of the brain adjacent to the tumor, to the gray as well as to the white substance, or attack the cerebral nerves. Now if, as has been observed above, the tumor need not cause such very pronounced symptoms in the place of its location, the consequence is that the local symptoms frequently may receive through the extensive neighborhood symptoms, something diffuse; but on the whole, the neighborhood symptoms indicate the location of the tumor, especially in the cases in which the tumor itself is situated in the so-called "silent" parts of the brain. From what direction this neighboring influence proceeds, can never be deduced from it alone. Neighboring disturbance of the motor area may have its origin quite as readily in a tumor of the frontal lobe as in one of the temporal lobe. Of especial importance as neighboring symptoms are also the disturbances in the domain of the cerebral nerves, as they appear very early in some cases.

In cases that deserve mention, although they are actually very rare, the pressure effects of a tumor may so manifest themselves, as to spare the directly neighboring areas, and affect the more distant ones. Thus, a tumor of the left posterior cranial fossa may injure mainly, or exclusively, by its pressure, the cerebral nerves of the right side. In like manner, the cases of homolateral hemiplegia, i. e., for instance, paralysis of the right side in tumor of the right hemisphere, are probably also to be explained in that, as a result of the displacement of the intracranial parts, under the pressure of the tumor, the opposite peduncle suffers most.

Excluding the area of extension, the local symptoms of cerebral tumor are qualitatively differentiated from the local symptoms of other cerebral diseases, in that they very requently lead to peculiarly severe irritation phenomena, as a result of the mechanically exciting effect of the new growth. These *irritation phenomena* are most striking in the realm of the motor zone, from which they then emanate in the form of epileptic attacks of *Jacksonian* type. Sensible and sensory irritation phenomena, furthermore, may be caused in the sphere of all the senses by irritation of their cortical representation. At the same time, there may be a change of phenomena of irritation and of those of functional loss. Irritation of the cerebral nerves themselves, which in the motor nerves manifest themselves in the form of muscular twitchings, in the sensory nerves as paræsthesias and pains, naturally occurs in the basal diseases.

Special Diagnostics and Symptomatology

In this section, we shall present the differential diagnosis, and then the local diagnostics of cerebral tumor.

The Diagnosis of "Tumor."—Corresponding to the division of tumor symptoms into local and general, two groups of patients may be differentiated: those in whom the general symptoms predominate or are present to the exclusion of other symptoms and those in whom the local symptoms either dominate the disease picture, or at least, are present in pronounced degree.

As has been shown, in the previous section, the general symptoms of tumor nearly all depend upon the increased brain pressure; therefore, in a large number of cases the main point is the *determination of brain pressure*, and in very case in which there is a suspicion of tumor, this point must be investigated. Of its resulting conditions, the most frequent is *headache*. The headache of tumor in its lighter degrees presents nothing characteristic, and even in its higher degrees it can never with certainty be distinguished, by its type, from headaches of other origin, for instance of migraine or uræmia. Hence this first rule, now probably followed generally by neurologists, to *examine ophthalmoscopically*, every individual, who presents himself with headache and, indeed, to so examine every sufferer from nervous disease. Considering the frequency of headache and the comparative rarity of tumor, one glance at the disc usually suffices to convince one that there is no neuritis optica, and this slight trouble is well repaid, since the absence of neuritis optica, even though not excluding absolutely the presence of a tumor, at least makes it highly improbable. The cases in which the headache is really the only tumor symptom and both choked disc and local symptoms are absent, can not be diagnosed at once.

The practical opthalmoscopic rule is to use, wherever possible, the instrument without having recourse to artificial dilatation of the pupils with atropin or homatropin. If the picture is doubtful, they naturally will have to be used; but before doing so the condition and the reaction of the pupils to light should be throughly studied. This is of decisive importance in several respects, especially for differential diagnosis in respect to syphilis and the effect of homatropin frequently lasts for a much longer time than physicians are generally accustomed to assume. Since, judging from our experience, the art of dependable investigation of the pupils is not wide spread, the neurologist, who is called in is sometimes unable in such cases to give a definite opinion, because of the preceding atropinisation.

Now if in a case a neuritis optica has been determined—it does not matter whether headache or some other symptom has led us to the determination—the developed choked disc, i. e., the prominence of the head of the optic nerve, occurs only in extremely rare cases from any other cause than increased brain pressure. But mere pallor and congestion in the retinal veins occurs more infrequently in multiple sclerosis, in encephalitis and finally it must be remembered, that simple chlorosis, multiple neuritis and uræmia may also produce pictures which can not be distinguished from those that are present in tumors. If the diagnosis can not be made in such cases from reasons that are to be found in the symptoms of these other diseases themselves, and it yet be essential, lumbar puncture is to be performed under the precautionary measures which will be mentioned in the section on treatment. This, however, will be obligatory only in a minority of cases, even though desirable in very many.

A neuritis optica, in which lumbar puncture does *not* demonstrate an increase in brain pressure (cf. above) must, in the highest degree, awake suspicion of one of the other causes of neuritis optica we have mentioned. It is at least certain, that in chlorosis, multiple sclerosis and multiple neuritis, there is no brain pressure; in uræmia it seems to be present occasionally but only transitorily.

The cases in which the most frequent and practically the most significant difficulties arise, are those in which the presence of brain pressure is established without any doubt, in which under conditions all the results of increased brain pressure mentioned in the preceding section are present, but in which no, or only very slight, local symptoms exist.

At first, it is naturally a matter of importance, to discover even the slightest signs of any local affection of the brain, because these even as neighboring symptoms, may give valuable indications as to the seat of the tumor. Most important here are naturally the symptoms of functional loss on the part of the motor region, or of the tracts going out from it, of which we shall mention here briefly only the Babinski toe reflex, Oppenheim's, Mendel-Bechterew's reflexes, Babinski's mouvement associé du tronc et de la cuisse, as well as the P. Marie's crossed adductor reflex, finally, unilateral absence of the abdominal or cremaster reflexes. Concerning the value of these symptoms for diagnosis, a former chapter must have treated. Frequently, only one of them is present, but every single one proves an affection of the motor tract and therefore must awaken suspicion of a cerebral tumor, which is just beginning to affect the region of the central convolutions, or to a basal tumor which is pressing upon the brain-stem, etc. Thus in many cases, at least, the side upon which the tumor is located may be diagnosed. It is self-evident that a careful inquiry must be made as to the occasional occurrence of Jacksonian convulsions. In important and urgent cases, it may be necessary, for purposes of observing these convulsive seizures, to place the patient in the permanent care of a trained nurse. Just as important as the symptoms which point to a disease of the motor tract are those which indicate an affection of a cerebral nerve, especially when they are unilateral. This concerns in particular, olfactory disturbances, pupillary differences, slight degrees of ptosis and that mild form of trigeminus affection, which manifested itself only by areflexia of the cornea. The cerebral nerve symptoms especially are adapted to awaken suspicion of a localization in one of the three cranial cavities.

These cases just mentioned, with but slightly pronounced focal symptoms, form only one group, which is practically especially difficult. For the more pronounced and massive the focal symptoms are, the more probable is the diagnosis of cerebral tumor from the first. But in every case the question must none the less be raised, as to whether there really exists a tumor or whether another disease, whose course may be associated with an increase in brain pressure, is the cause of the symptoms.

For the decision of this question, since we include gummata under the tumors, *meningitis serosa*, resp. *acquired hydrocephalus* are concerned especially in differential diagnosis. Only on rare occasions, as has been stated, can uræmia also cause an increase in pressure of some importance. Therefore, to decide between tumor and meningitis serosa, resp. hydrocephalus, in a large number of cases, recourse must be had to *the microscopic examination of the fluid withdrawn by lumbar puncture.* Here the finding of lymphocytosis speaks with great probability against the presence of a tumor, in which it is only found in extremely rare cases, far more, as especially Krönig emphasizes, for serous meningitis. Only in syphilitic tumors, lymphocytosis is usually present. The absence of lymphocytosis is not of the same importance, since cases of hydrocephalus with focal phenomena, but without lymphocytosis also occur, though it is probably found in the majority of cases. Besides, it must be mentioned, that in acquired hydrocephalus, the phenomena of the increased brain pressure may disappear and recur repeatedly over long periods of time; from the course of the disease, however, one can draw conclusions with any degree of certainty only in rare cases.

If in cases of meningitis serosa or hydrocephalus, focal symptoms are present, disease pictures may develop, which clinically can not be distinguished from tumor and which Nonne therefore designated as *pseudotumor cerebri.* The question of the origin of this hydrocephalus, which has not yet been satisfactorily answered—aside from the otitic causation, there is, in some cases, suspicion of syphilitic basis—is not to be discussed. It seems confusing, if those cases also, whose course is not accompanied by brain pressure, are counted, as some authors do, with this group. The local symptoms, which have been found to be most common in these cases, are, first, cerebellar phenomena, and secondly, motor irritation symptoms in the form of *Jacksonian* epilepsy. But others also, as, for instance, alternating paralysis, may occasionally be seen as symptoms of pseudotumor. The more pronounced the focal symptoms are, especially the phenomena of functional loss, the more chronic, and, at the same time, continuously progressive is the course of the disease; and the earlier and more decided the local symptoms appeared, the more probable is the diagnosis of genuine tumor, the more so, as at the same time, it must always be remembered, that the cases of pseudotumor are not, on the whole, frequent.

If it is impossible to form, even with the addition of lumbar puncture, a picture sufficiently clear from the local and general symptoms, then *Neisser-Pollack's cerebral puncture* must be tried with an attempt at aspiration of the tumor tissue; the positive result of this is naturally decisive, while the negative leaves doubts, since the puncture may not have struck exactly the site of the tumor. *Neisser-Pollack's* puncture is to be recommended, in case we are in doubt as to whether a *pachymeningitis hæmorrhagica* resp. a hæmatoma of the dura mater may not be present; here, by the aspiration of old blood, a decision can very frequently be made, and here with sufficient local diagnosis, even negative results are complete proof against the presence of a hæmatoma. But we have not, as yet, another absolutely certain means of differential diagnosis between tumor and pseudotumor. Only recovery, which is possible in pseudotumor, or the autopsy clears up in many cases, the nature of the disease.

A positive result from *Neisser's* puncture removes, at one stroke, together with the uncertainty of the diagnosis of tumor, the local diagnostic difficulty. But in most cases, since it should be performed only in a hospital, it is not at once available; its negative result is not convincing in many cases, and, at any rate, it should, in all cases, be only the last heroic means of local diagnosis. Though it can not be denied, that the introduction of cerebral puncture has caused some changes in local diagnosis, nothing could be more incorrect than to believe that it would be sufficient, unless one had previously exhausted all the other means of local diagnosis. But after the "diagnosis" tumor has once been made with a sufficient degree of probability, the central point of the diagnostic problem is still, as it was before, the attempt to localize the tumor exactly, according to its clinical symptoms.

But before we reach that point, we must discuss briefly the differential diagnosis of those tumors, which cause no increase in pressure and no choked disc. First of all it should be remembered that in certain locations a tumor but rarely causes brain pressure. This holds good particularly for pontile tumors. Nor need tumors of the cerebrum necessarily lead to brain pressure from the first. It is remarkable that cysticerci, and even those of the fourth ventricle, frequently run their course without choked disc. Occasionally, however, tumors in any location, even in the cerebellum, which as a rule, cause choked disc very early, run their course without it to the very end.

The cases of cerebral tumor, which run their whole course without brain pressure, especially, in particular without choked disc, but which have local symptoms, frequently rouse a suspicion of tumor because of the special state of consciousness and the violence of the headaches of the patient. But in every case of brain disease, which develops in younger persons, progressing, as it seems, gradually from the onset, a diagnosis of tumor must be considered. In by far the great majority of cases, the symptoms of tumor develop more slowly than those of all other (cerebral) diseases; this is especially true of *cerebral abscess, encephalitis, and pachymeningitis hæmorrhagica;* a *thrombotic softening* might sometimes create diagnostic difficulties in older persons. *Multiple sclerosis* may cause difficulties, first with regard to those cases which proceed for some time with stupor, headache and vomiting, and again with regard to distinguishing it from beginning pontile tumors, and from cerebellar tumors, which latter, however, rarely run their course without brain pressure. *Uræmia* may give rise to differential diagnostic pro's and con's in such cases, in which beside the headache and the stupor, convulsive attacks or hemiplegias develop, as they occur frequently in uræmia, and without any gross anatomic findings. The most important question in the differential diagnosis of cerebral tumor is naturally, always that relating to *syphilis*, if we classify the gummata among the cerebral tumors, that is, from vascular syphilis with its resulting conditions. Here the anamnesis and the discovery of syphilitic marks upon the body, or specific syphilitic symptoms of the nervous system, especially reflex pupillary rigidity, will, in most cases, make a determination possible at a relatively early period.

The probability of tumor always seems especially great in the presence of epileptic convulsions of *Jacksonian* type, which (cf. below) indicate a localization within the motor area. In these cases, beside the diseases already mentioned—we are speaking now only of the forms without brain pressure and choked disc—such forms of really *genuine epilepsy* must be thought of that in the beginning, and, in fact, for some time, may run their course under the picture of a localized *Jacksonian* epilepsy, without however the possibility of finding the least trace of a local change in the corresponding cortical center. Such cases have been operated on a number of times under the diagnosis, "tumor." Mistakes are best avoided in such cases by a careful anamnesis, which should be directed towards the discovery of characteristic signs of genuine epilepsy (heredity, attacks of petit-mal, etc.).

We shall return later to individual cases of differential diagnosis, in our discussion of the localization of tumor.

Confusion with *hysteria* should not occur. One should proceed from the standpoint that proof of the presence of hysteric symptoms does prove that hysteria, but not that *only* hysteria exists, that, moreover, a tumor should not be diagnosed without organic symptoms, but that the proof of a certain organic symptom excludes hysteria as the solitary cause of the disease.

Local Diagnosis of Tumor.—If now, independently of the state of the general diagnosis of tumor, we proceed to discuss the local diagnosis of tumor, we must first firmly establish a smaller group of local symptoms, by examination of the cranium. The careful *testing of the cranium* everywhere for tenderness to local tapping and pressure should never be forgotten. The establishment of this is by no means a certain sign, but in quite a number of cases it does, approximately, correspond to the site of the tumor. More frequently still, it corresponds at least to the side of the tumor. In many cases, there exists also at the point of the objective sensitiveness, the subjectively felt headache, which, however, is an uncertain symptom also. The objective local tenderness of the cranium may furnish a good test through the painful distortion of the face during the tapping, especially in patients in a stuporous condition. The local sensitiveness of the cranium to tapping and pressure affords more than a mere indication, only if it corresponds to the site, which upon other grounds is supposed to be the seat of the tumor; then it is a considerable support to the diagnosis.

Far rarer are certain phenomena to be established by *percussion of the cranium*, to which, following the example of English authors, *Bruns*, in Germany, has given special attention. One can, under certain conditions, ascertain in a circumscribed place, a change in the usually hollow percussion sound of the cranium in the sense of a tympanitic sound or a bruit de pot fêlé (cracked pot). They are probably the signs of a considerable thinning in the vault of the cranium, and they appear within the limits, in which such might possibly be caused by tumor. Since they occur therefore also in other processes, that attenuate the vault of the cranium, they can really be used diagnostically, only to support the diagnosis.

In some cases sounds in the vessels synchronous with the pulse, have been heard by auscultation, and not only, though usually, in aneurisms, but also in solid tumors, when these probably partly compress a vessel.

Tumors, which grow from the cranium, may appear on the external surface of the cranium itself.

The main group of local symptoms, which are evoked by the injury to the nerve tissue itself, may be treated here comparatively briefly, as the facts of cerebral localization have already been presented in a preceding chapter of this work.

It is to be noted at the outset that every local symptom has the more value, the more isolated and the more constant it is, and the earlier in the course of the disease it appears. That every local symptom, without exception, and especially if it appears in the later course of the disease, when a high degree of stupor already exists, may, in itself, be misleading, has been shown in considerable detail above. If, therefore, the patients are seen only in the later stages of the disease, it is always of great, sometimes of decisive importance, to become informed if possible, concerning the development and succession of the symptoms then present. It is helpful and extraordinarily important for the determination of the primary seat of a tumor, whether the anamnesis states that the patient has suffered off and on for six months from recurring spasms on the left side and that recently severe headaches and morbid drowsiness have appeared, or whether we discover that during the last six months the general symptoms of a cerebral disease have been manifest, and that now in the last week, a unilateral spasm has appeared once or twice. In the former case, we are inclined from the first to seek the tumor in the motor area itself, whereas in the latter, the spasms are to be regarded only as a neighborhood symptom, possibly of a tumor which is developing primarily in the frontal or temporal lobe. Diagnostic considerations of this sort, which must be inquired into in every case, can not, of course, be described in detail here.

Concerning *examination of the tumor patient*, one must not be frightened off by the stupor, but attempt after an objective investigation without the coöperation of the patient, in so far as this is possible to make the patient perform voluntary movements with the extremities (remembering the test for apraxia), with the eyes and speech by repeated demand upon him. One should try, if possible, a stereognostic examination with simple objects (key, knife, watch-chain, etc.). A test for hemianopsia also should be attempted in the simplest way; the patient, while looking at the examiner who is sitting opposite to him should be asked to catch hold of a handkerchief that is held up on one side. Occasionally stupefied patients can be roused a little, by taking them out of bed and putting them on a chair.

Now we shall consider the tumors of the cerebrum, progressing simply from the front backwards.

Frontal Lobe.—If the area, which lies in front of the precentral fissure, resp. of the anterior central convolution is referred to when the frontal lobe is spoken of, it is the more difficult to diagnose a tumor in this region, the more anteriorly it lies, but because of the location of the speech center on the left side, tumors there are far more easily recognized, than upon the right.

The theory of *Broca*, that the seat of the speech center is in the third left frontal convolution, has recently been disputed by *P. Marie* and it seems quite certain, that in a number of cases this motor center is situated not here, but farther back. For the practical localization of tumors of this region, however, the speech disturbances in the sense of motor aphasia, are, in every case significant (especially as disturbances in writing and reading may be included), particularly since the surgeon will never be satisfied with exposing only the base of the third frontal convolution, but will always expose a larger portion of the lobe. Stumbling over syllables, clumsiness in speech, etc., must always be considered as possible indications of motor aphasia.

Observations of tumor cases have not proven that the base of the second frontal convolution has a special importance for writing. On the other hand, this may really be the central point for the conjugated eye movements towards the opposite side. Stimulation of the corresponding area in animals causes their eyes to turn towards the opposite side, and in man also there are *Jacksonian* convulsions, which begin with the turning of the eyes and the head to the opposite side. These convulsions point to a localization of the tumor in this region, unless they are the beginning of a genuine epileptic paroxysm.

But if the epileptic convulsion limits itself exclusively to movements of the eyes and the head, or from here advances exactly in the form of a *Jacksonian* convulsion, and then, say, affects in addition only the face, one may to a certain extent be sure of the presence of an irritating morbid process, which is situated in the posterior portions of the frontal lobe. Déviation conjuguée as a symptom of functional loss on the side of the focus, plays but an unimportant rôle in the study of tumor localization; it is likewise only a transitory symptom in hemorrhages in this region. This probably results from the fact, that, first, the eye movement has, besides the center in the frontal field, a second one near the occipital pole in the brain, also that possibly with unilateral destruction the other side may act in a compensatory manner and that finally perhaps in all cases, where a pronounced déviation conjuguée does not exist, a more thorough investigation into this disturbance, in the sense of a difficulty in turning vision and the head, should be made, than usually now takes place.

Furthermore, in tumors of the frontal brain, *Moeli and Wernicke*, and specially Bruns, observed rather frequently a form of ataxia, which is supposed to correspond exactly with cerebellar ataxia, i. e., appears above all in walking and standing. This ataxia was especially pronounced in cases of bilateral disease of the frontal brain. In some cases of unilateral ataxia a deviation of gait in the direction of the diseased side has been observed just as in cerebellar tumors. By most authors this ataxia, in consideration of *Munk's* assertion that the frontal lobe subserves the movement of the trunk, was referred to the injury of this supposed sensory sphere of the trunk. This importance of the frontal brain for the trunk, as well as the theory of the very close connection between frontal brain and cerebellum seem very problematic to-day. On the other hand, it seems plausible, that an injury to the movements of the eyes and the head resp. nape of neck, which, as has been stated, indubitably have some connection with the frontal lobe, should lead to ataxia, especially if it be a bilateral disturbance. But it has by no means been proven, that this frontal brain ataxia is not a cerebellar ataxia, caused by the pressure with which a tumor of the frontal brain can push the cerebellum against the bony cranium. It is certain, that even in very large unilateral tumors of the frontal brain, ataxia may be wholly absent; but since it actually occurs in a large number of these cases, one will, whenever it is discovered consider the possibility of a tumor of the frontal brain in every instance.

There is great difference of opinion also as to the psychic disturbances in tumors of the frontal brain. After a number of investigators had expressed themselves that the frontal brain had special importance for the psychic functions, *Jastrowitz* described moria, idiocy, silliness with extraordinarily cheerful temperament, as characteristic of the tumors of the frontal lobe. *Oppenheim* speaks of a would-be facetiousness (Witzelsucht). As a matter of fact, it is scarcely questionable that a light hypomanic condition, a euphoria, with inclination to make would-be funny or silly remarks, occurs very frequently in tumors of the frontal brain. This euphoric mood stands in peculiar contrast to the slight stupor, by which dementia is only simulated. That this condition in the theoretic-physiological sense is a focal symptom of the frontal lobe, has become doubtful because it occurs likewise, though less frequently, in other locations. Even psychic disturbances, which were found to resemble progressive paralysis, have been observed in tumors of the frontal brain; but it is extremely infrequent, that there should be real difficulty as to the diagnosis between tumor and paralysis, because on the one hand, the choked disc as well as constant local symptoms of the projection paths belong to the greatest rarities in genuine progressive paralysis and because, on the other hand, the physical signs of the paralysis, especially reflex pupillary rigidity point almost only to the possibility of syphilitic tumors and almost certainly exclude genuine tumors.

The nearer the base a tumor of the frontal lobes is, the more are the olfactory as well as the optic nerves endangered. Unilateral olfactory disturbance may be of decisive importance for the local diagnosis, though, like every local symptom, it proves nothing *in itself*. At any rate, an olfactory test is never to be omitted. Unilateral blindness due to pressure upon the optic nerve, as well as unilateral choked disc occur also. Because the tumor presses forward and impinges upon the orbit, exophthalmus and disturbances of all the nerves that course here, may occur.

As cerebral neighboring symptoms there naturally occur motor symptoms as a result of the influence on the neighboring anterior central convolution resp. its corona radiation fibrillation; they may be either irritation phenomena or pareses.

Central convolutions and parietal lobes. Here we group together the region of the central convolutions and the parietal lobes, as in them are situated the centers for motion and sensation of the body. At the same time the line, to which the functionally so defined region extends backwards, is not sharply limited or at least not yet clearly decided. On the other hand, within this area a very sharp boundary is furnished by the fissure of *Rolando*. It may be regarded to-day as established, that genuine motor excitability belongs only to the region that is situated in front of the *Rolandic* fissure, in particular, to the anterior central convolution, as corresponds to the experiments with electrical stimulation of *Hitzig* and *Sherrington* in animals and of *F. Krause* and others in man. What holds good for electrical, certainly applies also for the mechanical excitability, such as may be brought into action by a tumor. The expression of this irritation is the localized epileptic (*Jacksonian*) convulsion and it is, as a matter of fact, very probable, that every *Jacksonian* convulsion is produced from the anterior central convolution, resp. from the paracentral lobe. But for practical localization this does not mean, that, if epileptic convulsions of Jacksonian type are present, the tumor must necessarily be situated in the anterior central convolution; on the contrary, very frequently, pressure from the posterior central convolution may be exerted on the anterior, so that *Jacksonian* convulsions result. But, as a matter of fact, it seems that the more circumscribed the irritation phenomena are and the more constant in their manner and extent, the nearer the tumor lies to the anterior central convolution. In no other place can tumors be so exactly localized as here, due to the detailed knowledge that we have of the situation of the excitable places in the anterior central convolution. We must remember that the motor centers for the most distal parts of the lower extremity lie farthest over towards the edge where two surfaces, supero-medial border, are in contact, that then laterally come the more proximal parts of the lower extremity, farther down the trunk, the upper extremities in proximal-distal direction, finally the face, then the muscles of chewing and of the tongue. There are *Jacksonian* convulsions, which are restricted almost to a single muscle, for instance, the extensor hallucis. They may spread for varying distances from their starting point; even to the other side. It is always of especial importance to determine the point, at which the convulsion begins. A *Jacksonian* attack may last far longer than an attack of genuine epilepsy, from which it is also distinguished by the retention of consciousness, a condition which is usual, when the area of extent is not too large.

The symptoms of functional loss of the anterior central convolution are the circumscribed pareses or paralyses, usually in the form of monoplegias, corresponding, in detail, to the seat of the tumor. In small tumors, the paralyses may be restricted to parts of the limb, as, for instance, the foot, even in rare cases to a toe, or, say, the second to the fifth finger. When the tumor is of greater extent, naturally complete hemiplegias may occur also.

The phenomena of functional loss may combine with the irritation phenomena; in the beginning of the disease, after *Jacksonian* convulsions, pareses may remain for a short time, which are explained by the exhaustion of the cortex. Later the pareses may be permanent, but occasional *Jacksonian* convulsions may always reappear from time to time in the paretic limbs. That in real cortical tumors of the motor area, irritation phenomena do not appear at all, is scarcely possible; such irritation phenomena may occur also in subcortical tumors, but here they are frequently lacking, especially in infiltrating tumors.

It is doubtful, whether in affections restricted to the anterior central convolution, any disturbances in sensation occur, with the exception, possibly of those of the deep sensation. At any rate, considerable disturbances in sensibility indicate an involvement of the convolutions situated behind the central fissure, resp. of the fibres emanating from them. If, in addition to a not too marked paresis, there is present a pronounced sensory disturbance—

destruction of the sense of touch, dulling of the sensations of pain, loss of the muscular sense—the tumor must be sought, behind the central convolution rather than in front of it. It seems as if the disturbances in sensibility occur especially then if, as the affection grows backwards, it reaches rather near the central fissure or possibly is restricted, on the whole, to the posterior central convolution. *Wernicke* in particular has called attention to the importance of the posterior central convolution for sensibility. In diseases of the parietal lobes, of the upper and lower parietal convolutions, *Oppenheim and Mills* have observed, predominantly, astereognosis, with only slight disturbances of the primary sensations. At any rate, it is very important, when tumor is suspected, to test the ability of recognizing objects by touch; if this is intact, one may be almost sure that no sensory disturbances whatever are present. If it is disturbed and no other severe sensory disturbances are present, suspicion turns to the parietal lobes, in the disease of which, it may be the only symptom. If no severe motor disturbances are present, then with severe sensory disturbances—not in pure astereognosis—paralysis of touch—ataxia is always present. This cerebral ataxia, however, is distinguished very readily from the spinal and cerebellar forms, in that the restlessness and wavering of the movements and also in the posture are very little manifested, as compared with the enormous mistakes made when directing and measuring the movements.

The sensory irritation phenomena of the area concerned in the disease have, up to this point, perhaps been given less attention than they deserve. Not so much the central pains, whose localization is not uniform, as the circumscribed paræsthesias indicate, that behind the central convolution and probably parallel to the motor area, the sensory area is organized analogous to the motor one. The paræsthesias especially, those which appear paroxysmally and spread precisely according to *Jacksonian* type, in the realm of the touch and temperature sensations, speak for this. Differentiation of the separate sensory qualities, one from the other, has not yet been successful.

With extensive tumor formations in the left senso-motor area, there appears, beside the paralysis of the right side, in right-handed persons *Liepmann's* apraxia of the left side, just as in foci of this region due to different causes. This symptom is also important for local diagnosis, in so far as it does not occur in tumors situated far below the cortex. Thus the sign of apraxia gains special significance in cases, in which there is apparently present an alterating paralysis of *Weber's* type, so that one is inclined to assume the presence of a tumor at the base of the cerebral peduncle. By the ascertainment of an apraxia, it may be made very probable, that the oculomotor disturbance is only a distant symptom due to pressure on the base of the middle cranial fossa.

The Temporal Lobe.—In the localization of tumors in the temporal lobes, there is also a great difference between the right and left side. A tumor on the left side can not be diagnosed without sensory or amnesic aphasia. A further localizing differentiation is practically scarcely possible and not necessary. In tumors of the right temporal lobe, since the functional loss does not cause severe disturbances, one has to depend chiefly upon the neighborhood symptoms, of which ptosis or mydriasis of the same side is especially important, as the result of pressure on the oculomotor trunk (*Wernicke*). Of other neighborhood symptoms, especially those concerned with the visual path, on the one hand, and the motor, on the other may appear. There are no unilateral auditory disturbances. Subjective auditory phenomena, buzzing, ringing in the ears, etc., have occasionally been observed as irritation symptoms. Disturbances of smell and taste, especially hallucinations in these realms seem to indicate that the tumor affects the medial parts of the temporal lobe (gyrus fornicatus, uncus).

Occipital Lobe.—The focal symptom of the occipital lobe, is hemianopsia situated on the side opposite that of the disease. Since a similar hemianopsia may occur, however, with the neoplasm located in any part of the course of the visual path from the tractus opticus, and this entire stretch is very frequently exposed to neighboring influences from the tumors of other regions (parietal and temporal lobes), it must be judged with care. A definitely ascertained hemianopic pupillary rigidity, would, according to *Wernicke*, speak for a disease of the tractus, that is, against the location of the tumor in the occipital lobe. Optic hallucinations in the hemianopic field of vision have several times been observed, as irritation phenomena from the occipital lobe. A tumor of the left occipital lobe may have as symptoms alexia, and (secondary) agraphia, especially if the substance of the gyrus angularis is affected. As neighboring symptoms in these tumors, which extend forward to the anterior part of the occipital lobe, are found most frequently symptoms of light sensory aphasia, especially difficulty in finding words. Occasionally, a tumor of the occipital lobe, may exert pressure backwards against the cerebellum and might thus lead to cerebellar symptoms.

Internal Capsule.—A tumor, which develops in the inner capsule, will be diagnosed, if the symptoms of a gradually developing hemiplegia are present, without severe irritation phenomena, especially when to such a hemiplegia still other symptoms, as, for instance, hemianopsia are added, in which case then the tumor will probably have to be located, where large parts of the fibres of the hemispheres lie closely together.

Ganglia of the Cerebrum.—Tumors in the caudate or lenticular nuclei, can not, as a rule, be diagnosed, but in any case may be suspected by the eventual neighboring influences, which make themselves felt especially in the internal capsule. Tumors of the thalamus may in typical cases cause unilateral sensory disturbances and hemianopsia; choreic twitchings and amimia have also been observed. Yet tumors of the thalamus have frequently been observed without any, or with only quite indefinite symptoms. On the other hand, the hemianæsthesia, and the visual disturbances, if present, may lead, obviously, to diagnostic difficulties, with respect to tumors of the hemisphere, and especially to those of the parietal lobe. As neighborhood symptoms in tumors of the thalamus, it is possible to observe motor disturbances through injury of the internal capsule, resp. the base of the cerebral peduncles, and oculomotor symptoms through involvement of the quadrigeminal bodies.

Corpus Callosum.—Diagnosis of the tumors of the corpus callosum has not been feasible until recently. After the recent investigations of *Liepmann*, there can be no doubt that the focal symptom of the corpus callosum, at least of its anterior part, is isolated motor apraxia of the left side, and instances of tumors of the corpus callosum have been published, which exhibited this symptom. Contrasted with this symptom, a supposedly very pronounced disturbance in intellect, which has been found in a number of cases, but is yet doubtful as a diagnostic sign, must unquestionably fall into the background. By the neighborhood influence of the tumor, or its encroachment from the corpus callosum upon the fibres of the hemisphere, pareses of one or both sides of the body will nearly always be combined with the unilateral apraxia, but nevertheless, in not too severe paretic disturbances, one can usually easily distinguish the apraxic components. But for a tumor of the posterior sections of the corpus callosum, we have, as yet, no certain sign.

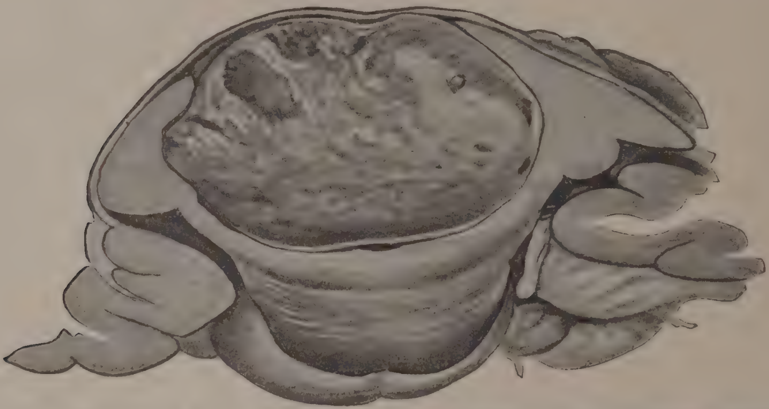

FIG. 170.—Teratoma of the quadrigeminal bodies. Collection of the pathological institution of the Friedrichshain hospital. (*L. Pick.*)

Brain Stem.—Tumors of the *quadrigeminal bodies* cause phenomena on the part of the muscles of the eye, through injury of the oculomotor nucleus, besides more or less pronounced motor and sensory disturbances through injury of the conducting tracts pressed together into a narrow space in this region. Pathognomonically, conjugated paralysis of the upward and downward movements of the eyes seem to be important for tumors in the quadrigeminal bodies (Fig. 170), the physiological basis of which is not yet clearly ascertained.

Tumors of the *pineal gland* may, if they grow downwards, cause the same symptoms as tumors of the quadrigeminal bodies.

The classic symptom of the tumors of the *base of the cerebral peduncle*, is upper alternating paralysis, oculomotor paralysis on one side, paralysis of the extremities on the other (*Weber's* type). The paresis of the extremities may be combined with a delicate tremor (*Benedict's* syndrome). Oculomotor paralysis with ataxia of the opposite side occurs in this region also—perhaps with peculiar frequency in an affection of the tegmentum, but also in that of the base. Because the tumors, especially the tuberculous growths, which show a predilection for this region, spread over both sides of the brain stem, very severe and complex bilateral disturbances may arise.

In a tumor of the *superior peduncles* of the cerebellum in this region, *Bonhöffer* has observed pronounced choreic symptoms.

Tumors of the *pons*, if they are unilateral, cause the well-known picture of the lower form of alternating paralysis, in which the facial of one side is most frequently paralyzed (*Gubler's* type), less frequently the abducens or trigeminus, and in addition a pyramidal lesion injures all the muscles on the opposite side, below this point in a manner characteristic of pyramidal disturbances. Furthermore disturbances in sensation may occur on the opposite side from the focus, which occasionally may be dissociated. Irritations of the nuclei of the cerebral nerves are also observed, and may lead, for instance, to twitchings in the facial region. As pathognomonically important as the paralysis of the vertical movements of the eye for quadrigeminal tumor, is the unilateral paralysis of the conjugated turning of vision to the side of the tumor in unilateral tumors if near the nucleus of the abducens. The narrow spacial relations that are found here, explain the fact, that the tumors of this region are not long confined to one side, and that the clinical picture then suffers corresponding changes.

Tumors, which develop within the *medulla oblongata*, are not common. Their symptomatology is easily deduced from the anatomy of this region. Local disturbances in the last three cerebral nerves, motor disturbances through injury of the pyramids, sensory disturbances through injury of the descending trigeminal root on the one hand, and the sensory tracts from the periphery of the body, on the other, are the most important phenomena.

Especially to be mentioned also is a miosis on the side of the neoplasm by interruption of the tracts, which lead to the sympathetic nucleus of origin in the dorsal portion of the spinal cord. It is obvious that the symptom complex closely resembles syringobulbia or can not be differentiated from it.

Cerebellum.—The cerebellum is especially frequently the seat of new growths. The main symptom of cerebellar tumor is the ataxia, which in its pronounced forms, gives the patient a gait characteristic of an intoxicated person, and which may be so severe that the patient can not take a step or even hold himself erect. It is further characteristic, that in spite of the patient's inability to balance himself, the ataxia is far less severe in the performance of movements, which, per se have nothing to do with the maintenance of the erect position of the body, resp. the co-ordination of the posture of the trunk. The patient, therefore, can execute the "heel to knee touching" or the "finger-nose" experiment, or carry a glass to his mouth fairly well, though in pronounced cases, never quite normally. Babinski has even called attention to the apparently paradoxical fact, that the cerebellar patient in certain positions—as, for instance, when he lies upon his back with his legs, which are bent at the knee, drawn up to the abdomen, by bending at the hips—may remain longer in this position than a normal person, and in direct contrast with the tabetic, in a veritably cataleptic manner. The general ataxia of high degree is found most frequently in large tumors, especially in those which begin in the vermis and affect both sides of the cerebellum.

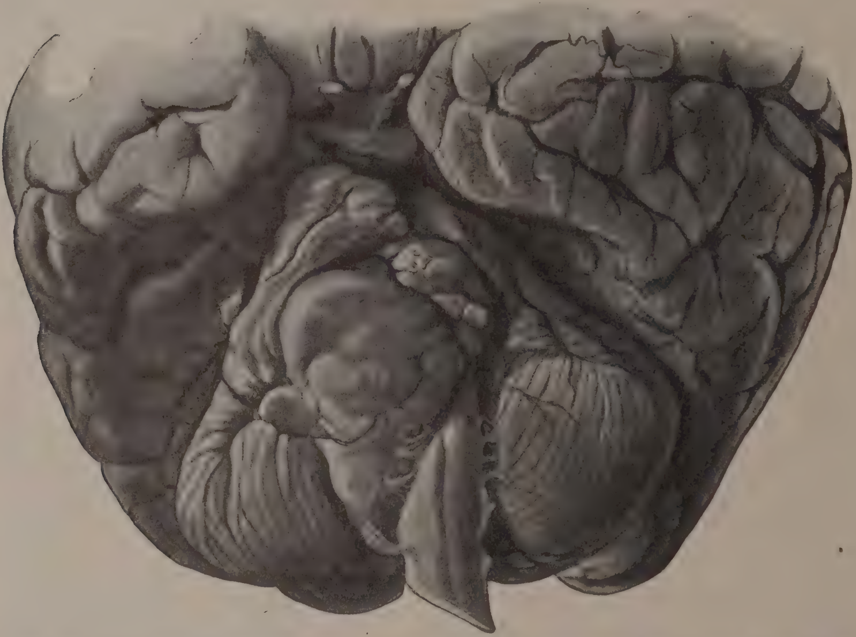

FIG. 171.—Glioma of the cerebellum. Collection of the pathological institution of the Friedrichshain hospital. (*L. Pick.*)

In the majority of cases it is also possible to determine the side of unilateral cerebellar tumors. Though it has been emphatically stated that tumors of the hemispheres of the cerebellum may run their course without producing any symptoms, one will be able to diagnose with a certain degree of probability, a unilateral cerebellar tumor, even when the symptoms are not very typical, if the diagnosis of tumor can be made at all. Of symptoms caused directly by the injury to the cerebellum, the following must be mentioned: the deviation toward the side of the tumor in walking, especially if one assigns to the patient the task of advancing straight to a definite goal with closed eyes. Secondly, the ataxia of the movement of the extremities, which, as has been noted, falls quantitatively far behind the marked disturbances in equilibrium of the large tumors, nevertheless in a unilateral tumor appears clearly, and then manifests itself on the side of the tumor. Thirdly, *Babinski* described as diadochokinesis the inability to perform movements in rapid succession, especially to supinate or pronate the hand quickly, one after the other. This disturbance, also, is found on the side of the tumor. Not unusual, furthermore, is a slight atony of the extremities on the same side; on the other hand, no marked sensory disturbances are found.

A main symptom of the cerebellar disease is vertigo, the genuine rotary vertigo. According to *Stewart* and *Holmes* the direction of this rotary vertigo is significant, in so far, as the subjectively perceived apparent movement of objects is always from the side of the tumor to the sound side. These tests, however, are often difficult to make, and the results probably do not in all cases coincide with the theory of the English writers.

Nystagmus is probably also a genuine cerebellar symptom and may, in unilateral affection, appear only when vision is turned to the diseased side.

Very important, in particular for the localization of cerebellar tumors, are the neighborhood symptoms. Preeminently here, stands *Oppenheim's* areflexia of the cornea, which occurs on the side of the tumor through the injury of the trigeminal trunk, and which only later may be followed by more pronounced disturbances in sensibility in the region of the trigeminus. Furthermore, abducens—facial—and hypoglossal pareses may occur through pressure on the nuclei or the nerve trunks. Pressure from the cerebellum on the brain stem may lead also to a pyramidal injury, unilateral or even bilateral. It is to be noted, that cases occur also, in which the pressure of the tumor is so directed, that it affects the cerebral nerves on the side opposite the seat of the tumor. In rare cases, when located anteriorly, cerebellar tumors may injure the quadrigeminal bodies and even the occipital lobe, and the phenomena corresponding to these regions obscure the picture of the cerebellar tumor.

Nuchal stiffness is not uncommon in cerebellar tumor and is probably of reflex causation due to the pressure upon the dura in the posterior cranial fossa. Unilateral affections frequently cause certain fixed postures of the head, which, in pronounced cases, offer insuperable resistance even to passive attempts at alteration.

A lsss common symptom is the peculiar tetanic convulsions of the body, described by *Jackson and Gowers*. It seems doubtful, whether hemiepileptic convulsions, which are described as being on the side of the tumor, are a true focal symptom of the cerebellum.

The tumors of the *fourth ventricle* resemble the cerebellar tumors in many respects. Through the pressure against the structures in the floor of the fourth ventricle, they cause phenomena in the cerebral nerves, and, furthermore, cerebellar phenomena of varying degrees may be present. Focal symptoms, however, may be altogether absent. Intense occipital headache, a rigidly held head, which is inclined forward, and a periodic course, possibly with intermissions of apparently perfect health, are characteristic of the majority of cases. In cysticercus of the fourth ventricle, which, in the main, produces quite the same symptoms as a solid tumor, the appearance of violent cerebral phenomena has been observed several times—vertigo, vomiting, falling to the ground—after a sudden active or passive change in the position of the head, *Bruns'* symptom, that must probably be traced back to a change in the location of the movable cysticercus.

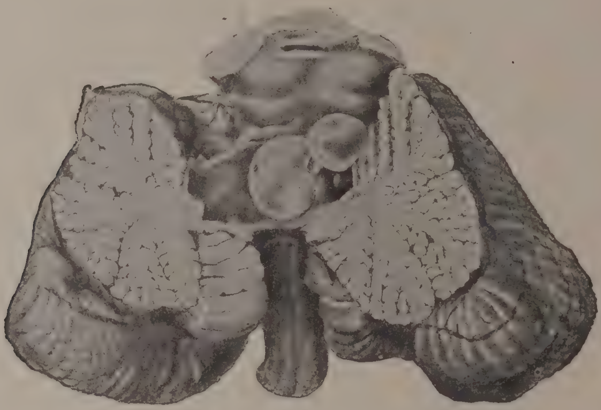

FIG. 172.—Cysticercus racemosus of the fourth ventricle from A. Stern. Collection of the pathological institution of the Friedrichshain Hospital. (*L. Pick.*)

In tumors of the cerebellum and of the fourth ventricle, death often occurs suddenly as a result of paralysis of the respiratory center. In the same way, all the general phenomena, which appear through the influence of the cerebral pressure on the centers of the medulla oblongata, are especially pronounced in tumors of the posterior cranial group because of their local influence.

Tumors of the Base.—The tumors, which begin at the base, originating from the bones or the cerebral membranes, because of their location, are distinguished in that they first, and sometimes for a long time afterwards, affect the cerebral nerves exclusively. Of greater frequency, and exhibiting typical symptoms are especially two localizations of basal tumors—one the hypophysis tumor, the other the acusticus tumor (tumor of the auditory nerve).

The focal symptom of *tumor of the hypophysis* is bitemporal hemianopsia, which originates from pressure on the chiasm. The connection of tumors of the hypophysis with acromegaly will be discussed elsewhere. Especially remarkable in tumors of the hypophysis are the frequently observed general disturbances, especially obesity and amenorrhœa.

Tumor of the hypophysis, moreover, is the only localization of brain tumors in which the investigation with X-rays frequently furthers the diagnosis, since, as *Oppenheim* first demonstrated, it may disclose a bulging and widening of the sella turcica due to the tumor.

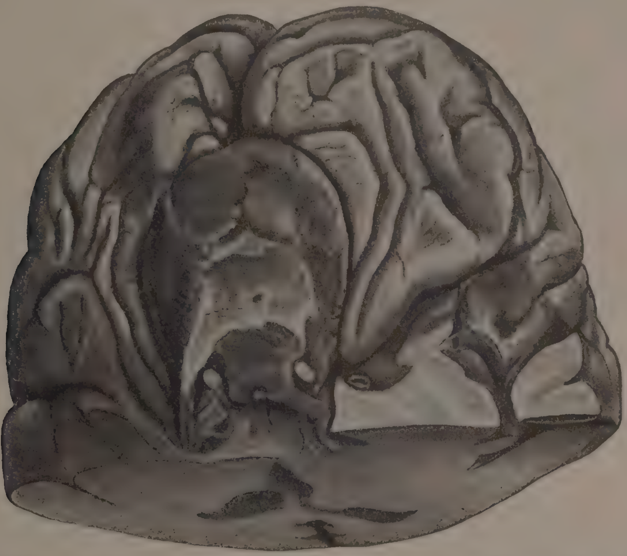

FIG. 173.—Adenoma of the hypophysis. Collection of the pathological institution of the Friedrichshain Hospital. (*L. Pick.*)

The comparatively frequent tumors in the intra-cranial part of the *acusticus*, usually develop a typical symptom-complex, which is founded upon their situation in the cerebellopontine angle. They probably always begin with unilateral auditory disturbances, which may for a long time (subjectively) escape the notice of the patient himself, sometimes until the other phenomena appear. Objectively, one discovers very severe auditory disturbances, or complete deafness, while the ear-findings are normal. In cases, in which there is simultaneous chronic inflammation of the middle ear, the determination of the character of auditory disturbance may make difficulties. Vestibular disturbances occur also, sometimes in the form of paroxysmal attacks (*Ziehen*). The total or almost total intactness of the facial nerve, which lies directly adjacent to the auditory, is frequent and very striking. Furthermore, there then appear cerebellar disturbances, and by compression of the pons, phenomena of contralateral paralysis of the limbs and occasionally ocular palsies "looking" paralysis (Blicklaehmung) towards the side of the tumor. These acusticus tumors are distinguished from the intrapontine tumors, first by the fact that in them choked disc is usually very pronounced, whereas it is usually absent in the intrapontine tumors, and that auditory disturbances of such high degree as usually occur in acusticus tumors are very rare indeed in the former. Acusticus tumors have also been observed to occur bilaterally in a considerable number of cases.

The other tumors of the base produce phenomena, which correspond to the cerebral nerves that are thereby affected. For instance, a trigeminal neuralgia might be produced by a tumor of the middle cranial group. Basal tumors, which, by creeping along the base, affect a whole series of cerebral nerves, are not so infrequent.

Multiple tumors. Multiple tumors can generally not be diagnosed. They may be suspected, if early in the disease symptoms appear, which can not be explained by the presence of a single focus. It is sufficiently clear from what has been said, that, in the later development of a single tumor, the presence of two foci may be simulated by the local symptoms of brain pressure. The principle will therefore always be to trace all phenomena if possible back to one focus.

Lumbar and Cerebral Puncture.—To complete, and, in a certain sense, to summarize what has already been said, at some length regarding the use of lumbar and cerebral puncture, we would add the following:

Lumbar puncture is justified as a diagnostic method, when a tumor is suspected, if it is performed with the necessary caution. By necessary precaution is meant the continuous measuring of the pressure during the withdrawal of the fluid which, to prevent its accidental loss, is best done with *Koenig's* apparatus. The pressure should be lowered but slowly, and should not fall below 120 mm. of water. If the pressure should be very high at first, it is advisable to keep it above this limit. Sudden marked decrease in pressure occurs in tumors of the posterior cranial fossa (cf. p. 603) and is a sign that withdrawal should be stopped at once. It is absolutely wrong, especially when there is a suspicion of tumor, to permit the cerebro-spinal fluid simply to run off and to guess at the degree of pressure by the force of the flow. The consequences of careless withdrawal may be hemorrhages from the tumor or even from the vessels of the base of the brain, which may cause death. Furthermore, death may occur after a lumbar puncture if by the lowering of the pressure of the spinal canal as the afterflow is cut off from the cerebral ventricles, the brain is to a certain extent sucked fast and the cerebellum and the medulla oblongata are pressed against the foramen magnum. Therefore, especial care is necessary in cases where the pressure suddenly relaxes. Certainly, some isolated accidents will occasionally happen, even if lumbar puncture is managed with the utmost care, and it is certainly not to be regarded as an absolutely matter of course operation; but considering its great usefulness, these slight dangers, which are not lacking in any method, are completely lost sight of. The worst that can be said in most cases of cerebral tumor, where death should be laid at the door of lumbar puncture, is that the unavoidable end has only been somewhat hastened.

Neisser-Pollack's cerebral puncture which after synchronous perforation of the skin and the cranial bone with fine electric drills by means of fine needles, aspirates the cerebral, or the tumor substance, is likely to be more dangerous than lumbar puncture. The danger lies here in direct injury to the vessels of the brain membranes by the needle; more or less severe hemorrhages beneath the meninges have occurred repeatedly. The possibility must also be reckoned with that occasionally an angioma or an aneurism might be pierced. To pierce the skull at haphazard in a numer of places until one had or had not found the tumor, in cases where tumor of uncertain location is only suspected should never be attempted. Under no circumstances should even a single puncture be regarded as an indifferent operation, relative to its effects upon the functions of the brain. The Neisser-Pollack puncture is probably of service where clinical observation hesitates between two localizations, for instance, frontal brain and cerebellum. It should be especially easy to decide on the puncture of silent parts of the brain. A negative result, moreover, will never wholly disprove the presence of a tumor in the vicinity of the puncture, since one can not puncture everywhere. A special importance might, as has been mentioned, accrue to cerebral puncture in distinguishing tumors from hemorrhages in doubtful cases.

Diagnosis of the Type of Tumor.—If a tumor is once localized, the determination of its type becomes of importance also. Here we are not taking into consideration Neisser's puncture, which, in case of positive result, clears up the histological structure of the tumor also. In addition it should also be only remarked, that cerebral puncture is of especial service in the differential diagnosis from cysts. A part from all this however the typical location of some sorts of tumors lends a certain amount of probability as to the presence of definite structures. Thus, acusticus tumors are always fibromata; tumors of the brain stem, in children usually tubercles; further tumors of the pons, usually gliomata; basal cerebral nerve paralyses always cast suspicion first on gummata. Tumors of the hypophysis are mostly adenomata. Tumors of the fourth ventricle will always suggest a cysticercus.

The distinction between syphilitic and non-syphilitic new formation is practically of the greatest importance. The possibility of syphilitic cerebral tumor will always in the first place be thought of too if there is syphilis at all in the history or if some syphilitic signs are to be found upon the body; or finally, if reflex pupillary rigidity is present. Lymphocytosis of the spinal fluid occurs, presupposing the presence of tumor, almost exclusively in syphilitic disease, but may be absent also in occasional cases. In gummatous meningitis, it is probably present without any exception. Furthermore, we now have in the *Wassermann* serum reaction a method, of which the positive result determines with certainty, that syphilis has once been present, or still exists, from which, however, it is not to be concluded absolutely, that the cerebral disease in question is also syphilitic, just as the negative result does not with certainty exclude syphilis. The performance of the reaction on the cerebro-spinal fluid, instead of the blood serum does not seem, to offer any advantages in the case of the syphilitic cerebral diseases.

The probability of tubercular new formations will, in analogous manner, as in syphilis, have to be supported on the simultaneous occurrence of other manifestations of tuberculosis in the body.

Differential diagnosis, between the true tumors is usually impossible, with exception of the metastatic growths, in which the diagnosis is furnished by the primary tumor.

Treatment

The only method, which can lead to a cure in genuine cerebral tumor, is an operation. Since *Wernicke* established the idea of operative treatment of cerebral tumors and *Horsley* first successfully operated upon one, the operation has been attempted in hundreds of cases; failures and successes are registered, and the question as to the indications for the operation has been treated from all standpoints. That exaggerated expectations could not be fulfilled, is due not only to the difficulty in diagnosis, but still more to the fact, that very many cerebral tumors are located in inaccessible regions of the brain, or, because of their histological structure, or their inextricable connection with parts that must not be touched, can not be wholly removed. It is also true, that opinions as to the operative accessibility of some parts of the brain have changed; the posterior cranial fossa, which formerly represented a noli me tangere, is to-day, especially because of the cerebellar tumors, a place often and in some cases, successfully attempted by surgeons. Indeed, even tumors of the hypophysis have been attacked from the nose, though, it is true, without permanent result. From the convexity of the cerebrum tumors may be removed without difficulty; tumors of the radiation of medullary fibres are also operable, and one tumor has even been removed from the thalamus. Tumors of the corpus callosum may certainly be operatively attacked. Surgically, the brain stem and parts of the base (cf., f. i., the tumor of Fig. 168) are not to be touched. In fact of the basal tumors, only the acusticus tumor offers any chances. Operation is indicated when a genuine tumor can be diagnosed from the standpoint of its locality, and this is a place surgically attainable. *Bruns* figures that this is possible, i. e., thus far has been, in 35% of the cases. The dangers of the operations in themselves alone, and the impossibility of completely operating not only upon infiltrating tumors, but in many cases, upon the well defined tumors also, make the results far less favorable. *Bruns* reckons according to his own statistics and those of others only 9% of clinically diagnosed, and at the same time, technically completely removable tumors, in which extirpation was feasible; and finally, according to *Oppenheim*, of 75 cases operated on by him, only in 5 cases = 7% lasting, complete success, in 5 other cases = 7% cure with defects was attained. Since the cases operated upon, as about coincides with *Brun's* statistics, are at most one third—rather less than more—of the cases under medical observation, which should be diagnosed as tumor, we would have scarcely more than 2% of completely cured cases of cerebral tumor, and 2% more, in whom defects remain after operations. This is certainly a small percentage, but must not discourage us from proceeding along the operative pathway. It is self-evident that the patient, and his relatives, must be informed as to the dangers of the operation. The operation may be completely successful, when, after extirpation of a rapidly growing tumor the substance of the brain, which has been injured by the pressure, is totally restored, and even the general symptoms entirely disappear. But, on the other hand, permanent defects of local character may remain, if the tumor has irreparably injured the substance of the brain, or if the cicatrix leads to a permanent injury. A neuritic atrophy of the optic nerves especially, never retrogresses. Recurrences appear, though not very frequently, after circumscribed tumors have apparently been completely removed. Gliomata can scarcely ever be extirpated completely. The operative treatment of simple cysts, which is not included in the above statistics, naturally presents much better results. Tubercles also were removed by operation in a number of cases with permanent success. Occasionally, also a single cysticercus, or echinococcus has been met with but usually they are multiple. The idea of operating upon cysticercus of the fourth ventricle has not as yet been put into execution. Metastatic tumors, when recognized as such, are not to be operated upon.

The syphilitic tumors, likewise, are, as a rule, not to be operated upon, but are to be treated with mercury and iodide of potassium and, if possible, with large doses, in one of the customary methods. In most cases, treatment by inunction is better endured than by injection, especially if the insoluble salts are used and is equally effective. But it may happen that the operative removal of even syphilitic tumors is indicated; for, in the first place, cases are not infrequently reported in which even gummata either do not react at all or quite insufficiently to energetic and continued treatment with Hg. and iodides; in such cases the expediency of operation must depend upon the severity of the case. Or it also may happen, that there exists an intolerance to Hg. or that the patient comes too late for medical treatment and that one can no longer wait for the result of treatment by Hg., since there is imminent danger of total blindness within a few days.

In general, with every cerebral tumor, antisyphilitic treatment is to be tried first, and rightly too, with the idea that it might be syphilis. But it is noteworthy, that tumors of all sorts very frequently react to this treatment with considerable temporary improvement, even when they are not due to syphilis, so that too much time should not be wasted in an attempt at internal treatment, if syphilis is improbable. If the tumor can not be localized, or can not be extirpated, palliative (decompressive) trepanation—making an opening in the skull—should be considered in a number of cases. There is no doubt that in many cases, it causes considerable improvement in the general symptoms, that it returns individuals, who have been gravely ill, to life and work for months and years, and that it can above all, prevent the development and aggravation of choked disc. But such results can not be promised, since in some cases even this procedure is absolutely of no value.

If palliative trepanation is refused, repeated lumbar punctures may be very helpful and bring relief, especially in those cases, in which the brain pressure returns but slowly and it suffices therefore, to make a puncture once in every few weeks.

In syphilitic cases, in which mercury and iodide have failed, intravascular injection of salvarsan or neo-salvarsan is justifiable. Mercury and iodide should be given between and after the injections (*Burr*).

In inoperable cases, arsenic in large doses, should by all means be tried.

For the headaches, the nervina are to be employed, even though only of symptomatic value, but in severe cases, only morphine helps.

The **prognosis** is evident from what has been said about the course of the various forms or tumor-like diseases, and furthermore, about the possibilities of treatment.

4. ABSCESS OF THE BRAIN

BY

M. LEWANDOWSKY (Berlin)

In contradistinction to the non purulent encephalitis, which is discussed in the next chapter, the purulent encephalitis—the cerebral abscess—is a uniform and well defined form of disease.

Etiology

The direct cause of the origin of cerebral abscess is always the penetration of *pusproducing micro-organisms* into the substance of the brain. Aseptic suppurations can be artificially produced, by experiment, in the brain as well as in other parts of the body; this artificial possibility however is out of the question in the disease, cerebral abscess. But on the other hand, it seems, as if the same micro-organisms, which at one time produce a cerebral abscess at another may lead only to a non purulent inflammation, especially to an encephalitis hemorrhagica. This is true for a number of bacteria, as for instance for the influenza bacillus and the pneumococcus. Nothing is yet known about the peculiar conditions, in which, under the influence of these microbes, at one time the non-purulent, at another the purulent encephalitis occurs.

In the great majority of cases of cerebral abscess the pus-producing micro-organisms found are the ordinary pyogenic micro-organisms, the streptococci and staphylococci. Among others there are found also the bacilli of influenza and of typhoid, the pneumococcus, *Friedländer's* pneumonia bacillus, *Weichselbaum's* diplococcus. That the tubercle bacillus also can cause a true abscess, which is not the result of a solitary tubercle breaking down and becoming an abscess, has been asserted by *A. Fränkel* and *Nonne*. In rare cases the streptothrix varieties, finally also the oidium albicans and actinomyces are found.

These pus-producing micro-organisms reach the brain substance either by way of the general circulation through the blood or they enter the brain directly from its vicinity by contiguity. The much discussed question, as to whether there is an *idiopathic cerebral abscess*, is, at bottom, very unimportant. It would mean only, whether pus organisms could settle in the brain, without having previously caused suppuration in another place. Theoretically this is as possible, as it is practically difficult to determine in the individual case. For we may have quite forgotten the primary purulent infection, possibly an unimportant whitlow, or a slight completely resolved otitis media, when the abscess appears, or perhaps it may not have been observed at all. An observation by *Strümpell* occupies a special position in this question; he observed four cases of cerebral abscess during an epidemic of meningitis cerebrospinalis epidemica. But perhaps these, too, were not idiopathic abscess, since here the micro-organisms were brought to the brain substance from the cerebro spinal fluid, even though it is possible that the signs of the meningitis itself had disappeared long before the time of the discovery of the abscess. At any rate, this fact must be remembered, that even in the most careful postmortem examination, excepting the cerebral abscess, occasionally no sign of an existing or preceding suppuration in the body can be found.

The method of origin of *metastatic cerebral abscess*, is the same as that of metastatic suppuration in other parts of the body. Suppuration in any part of the body, a phlegmon of the arm, suppuration of a joint, purulent peritonitis, purulent angina, a carbuncle, etc., can, by carrying the purulent matter to the brain, occasionally lead to the formation of a cerebral abscess.

Likewise, abscesses are occasionally formed in the brain, as in other places, as a result of the infectious diseases, scarlet fever, measles, erysipelas, influenza, typhoid. Cerebral abscess may also, together with formations of abscesses in other organs, appear as a partial phenomenon of a pyæmia. Only one kind of production of the metastatic cerebral abscess is of special frequency and importance, viz. that which results from *purulent diseases of the thoracic cavity*, a connection the frequency of which *Virchow* was the first to recognize. The brain is most endangered by purulent bronchitis, especially when bronchiectases are present; empyema pulmonum, gangrene of the lungs, abscess of the lungs follow, but they are considerably less dangerous. The path of purulent metastasis is very short in pulmonary diseases, since the infectious material reaches the left side of the heart directly from the lung and thence finds its way into the main circulation of the body. But it has by no means been fully explained, why under these conditions, the brain is seemingly the seat of the metastasis so much more frequently than other parts of the body. In the brain itself, the area supplied by the art. fossæ Sylvii, especially by the left branch, is the favorite seat of the metastatic abscess. It is noteworthy, that even the metastatic abscesses are not always multiple, but are very frequently—according to *Gowers*, in half the cases—solitary.

Far more important than all these metastatic cerebral abscesses is the *second group*, which consists of those abscesses in which the micro-organisms reach the brain more directly. This group falls into two subordinate groups, one which owes its origin to traumatism, the other is based upon the effusion into the brain of purulent matter present in its immediate vicinity.

The simplest manner, in which *traumatic cerebral abscesses* may develop, is, by the direct introduction into the brain through the cranial injury of the pus-forming organisms, as through a puncture or a gunshot wound. At the same time the larger foreign body, to which the micro-organisms were attached, that is the blade of the knife, or even hairs from the scalp, may remain in the brain itself, but may, of course, have also been removed. In the majority of cases, however, it is not at all a matter of the intrusion of actual, relatively large foreign bodies, but it is quite self-evident, that perforating cranial wounds, complicated fractures of the skull, etc., always afford an opportunity for the introduction of micro-organisms. Thus in some cases the external cranial wound may apparently have healed aseptically, while in other cases a suppuration of the bone, is followed secondarily by a cerebral abscess.

But it is not at all necessary, that the cranium should be injured for a cerebral abscess to supervene. Micro-organisms are apparently able to reach the brain from the infected wound in the soft parts through the skull and the cerebral membranes. Also in these cases, the abscess is nearly always found at the site of the injury, but there are rare cases in which the abscess is said to have developed at the site of the contre-coup (*Gowers*).

There are several possible ways in which, even without any injury to the cranium, the pyogenic micro-organisms may reach the brain from the covering of the skull. A thrombosis may establish itself in the venous vessels, which course through the cranium, and thus the infectious process is either directly transferred to the contents of the skull, or by retrogressive transportation, without intermediate thrombosis, infectious material may be carried into the brain.

Apart from the traumatic cerebral abscesses, *otitis* is by far the most important cause of cerebral abscess, in 90% of the cases the *chronic form of otitis media purulenta*, in 10% the *acute form*. At the earliest, a manifest abscess was found three weeks after the beginning of an acute suppuration of the ear, whereas in the cases of chronic otitis this may have existed for 30–40 years and even longer. In spite of the comparative frequency of cerebral abscesses in otitis, it is, nevertheless, considering the number of cases of otitis, a very rare disease. In 1137 cases of otitis media purulenta, there were found according to *Chauvel*, only two cases of cerebral abscess. The cholesteatoma of the petrous portion of the temporal bone is especially dangerous. The statistics given by authors, as to what percentage of the purulent intra-cranial sequelæ of otitis media the cerebral abscesses form, fluctuate within very wide limits. In any case, the extra-dural suppuration is far more frequent and probably also meningitis and sinus thrombosis.

The otitic cerebral abscesses are usually brought about by a caries of the temporal bone, and the usual starting points are the tegmen tympani and the roof of the antrum mastoideum. Associated with the cerebral abscess, there is often found also an extra-dural suppuration. The cerebellar abscess has its origin either from the cells of the mastoid process in the fossa sigmoidea, or from the labyrinth of the ear, which as *Jansen* first showed, is frequently diseased. In addition to the direct transmission of the pus from a caries, there is also to be considered the conduction through natural pathways, as the spaces between the bones, the petrosquamus fissure, the internal auditory meatus, the vestibular aquæduct. The otitic abscesses are found almost exclusively in the cerebellum or in the temporal lobes, and it is the otitic diseases in the region of the middle cranial fossa, which lead to abscesses of the temporal lobes, while those in the posterior lead to abscesses of the cerebellum. The abscesses of the temporal lobe develop usually in the convolutions adjacent to the petrous bone, the third temporal convolution, and the gyrus fusiformis. *Wernicke's* center is usually spared.

In the cerebellum, the abscess usually lies in the lateral anterior part of the hemisphere.

The abscesses may be connected with the meninges, or with the primary focus in the bone, by a fistula. But even when this is not the case, the cortex is usually diseased. In some cases, however, even with the most minute examination, no direct connection could be found between suppuration of the ear and cerebral abscess, and this can not be wondered at since in traumatic abscesses also, as has just been mentioned, a direct connection with the injury, that is the place where the micro-organisms penetrated, can not always be demonstrated, but the transmission must be supposed to have been by way of the vessels.

Far less frequent than the otitic, are the *rhinogenic* cerebral abscesses. Here too it is nearly always a caries of the bone, either of the osseous wall of the sinus frontalis, or of the ethmoid bone, etc., which is the connecting link. The rhinogenic abscesses are found almost exclusively in the frontal brain.

Pathological Anatomy

An abscess of the brain may attain extraordinary size (Fig. 174); it may occupy almost an entire hemisphere. On the other hand, some are found no larger than the size of a pin's head. At the autopsy, if the abscess has caused death, one finds most frequently abscesses in the cerebrum of the size of hens' eggs or apples; in the cerebellum, they are smaller in size. From the living patient amounts up to 400 g. of pus have been evacuated.

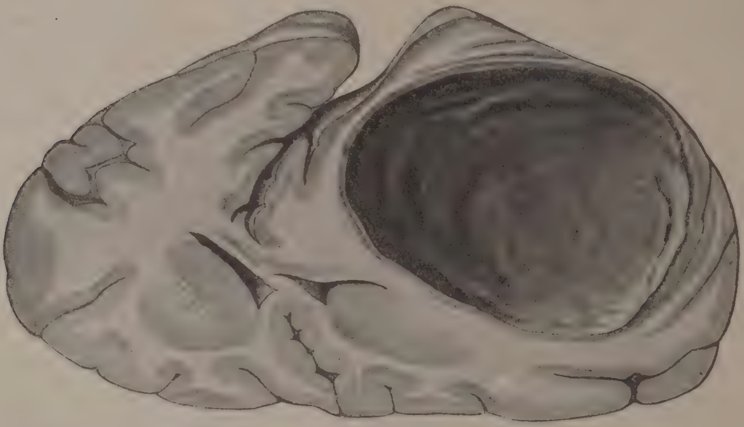

Fig. 174.—Very large abscess of the right hemisphere. Collection of the pathological institution of the Friedrichshain Hospital. (*L. Pick.*)

The pus is usually green or greenish yellow, usually has no odor, is only rarely offensive; it is mostly of thin fluid consistency.

The form of the abscess is at first irregular (Fig. 176); with the formation of its membrane, it usually develops into a regularly round or ovoid cavity (Fig. 174). But irregular cavities, divided by partitions into several chambers, occur also not infrequently (Fig. 175).

There is no reason why the *histogenesis* of brain abscess should be supposed to differ from that of abscesses in other organs. The purulent inflammation is everywhere the prototype of the inflammation, the characteristic of which is the migration of polynuclear leucocytes from the vessels. Under the influence of this extensive leucocyte exodus, a quick breaking down of the nervous tissue takes place, which at first seems softened and edematous. The changes in the glia are quite secondary. The immediate vicinity of the abscess cavity is frequently made up of a zone of softened tissue (Fig. 175). But it is incorrect as *Wernicke* has emphasized, at least, for the majority of cases, to suppose that a hemorrhagic encephalitis is a necessary antecedent stage of the abscess. It is even doubtful whether hemorrhagic encephalitis can become transformed into an abscess at all.

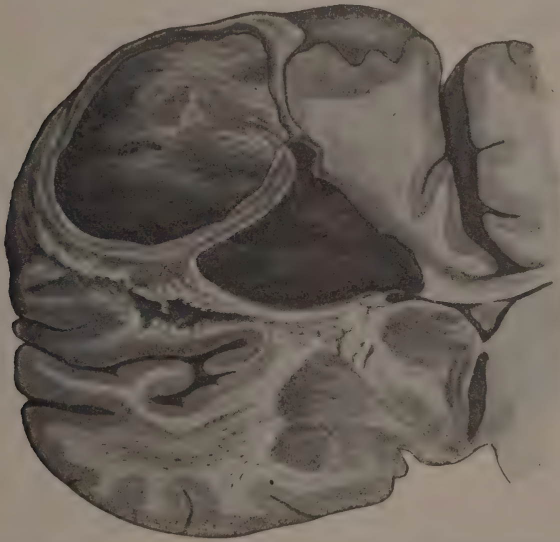

FIG. 175.—Abscess with two cavities. Collection of the pathological institution of the Friedrichshain Hospital. (*L. Pick.*)

In all abscesses of longer standing, there is formed around the abscess a *membrane*, the first indication of which may appear, according to the experimental investigations of *Friedmann* as early as the 5–6th day, and according to an observation of *Cassirer*, in man also as early as the 8–10th day. This membrane is formed of the connective tissue of the bloodvessels, seems therefore to be of purely mesodermal origin. It may assume the form of a strong rind, so that the entire abscess can sometimes be peeled away with this rind from the brain. The soft cerebral membranes may fuse with the membrane of the abscess. This covering, however, furnishes protection to the vicinity from bacterial invasion only to a very limited extent even though the long latency of some abscesses can be explained only by their long exclusion by means of the membrane. The large abscesses, acting like tumors, may cause considerable displacement and crowding out of parts of the brain (Fig. 174) and lead to the development of hydrocephalus by increase in cerebro-spinal pressure. Nor can the mechanism of this increase in pressure be any other than that discussed in the section on tumor (p. 602). The otitic cerebral abscess is frequently combined with sinus-thrombosis.

Those changes, which give rise to the formation of an abscess, have already been mentioned. The abscess finally leads, as a rule, to perforation either into the ventricles or into the subarachnoid space, and thereby to the development of a purulent meningitis. In otitic abscess, a meningitis may result also through direct infection of the meninges as a result of the primary disease. But even in the metastatic abscess, one finds, in spite of existing meningitis, not always an actual place of penetration, so that it is necessary to assume the possibility of the origin of meningitis by the microbes being carried from the abscess through pre-formed pathways.

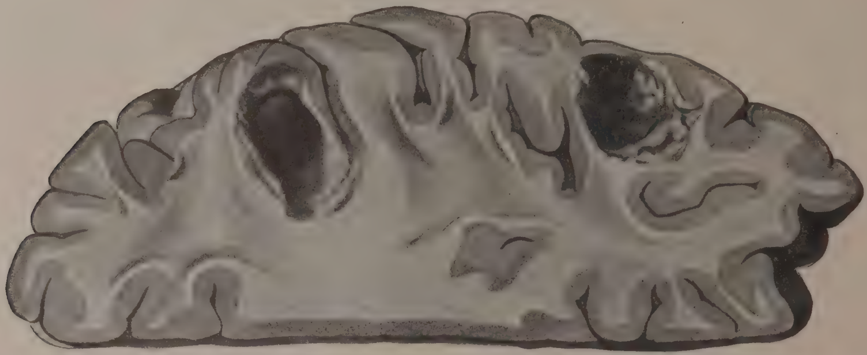

Fig. 176.—Multiple abscesses. Collection of the pathological institution of the Friedrichshain Hospital. (*L. Pick.*)

On the other hand there may also be a connection, ascertainable, even during the life of the patient between the cavity of the abscess and the various divisions of the ear.

General Symptomatology

Abscess of the brain may run its course in a few days, but in some cases it may remain latent for years and even decades. The traumatic superficial cortical abscesses may terminate fatally very rapidly, particularly if, as frequently happens, they are connected almost from the very first, with traumatic meningitis. Then only a few days need elapse between the effect of the traumatism and the fatal end. In such cases we are dealing with a disease, which advances evenly and rapidly from the beginning, from the moment of infection.

In the other cases, *three stages* are usually differentiated: 1, the initial stage; 2, that of latency, and 3, that of activity, extending possibly to the fatal termination. This division is, to this extent, not very strict, in that in the first place, the initial stage may be altogether lacking, or at least appear wholly without symptoms; in the second place, as in the above mentioned group of early traumatic abscesses, there may be no stage of latency and, in the third, even the stage of manifestation of the disease may be lacking since death may occur quite suddenly in a condition of apparent health.

The *initial stage*, if it be present, is characterized by the signs of a febrile disease, with cerebral symptoms, headache, vomiting, stupor. Naturally there are no exact clinical observations, as to what pathologic-anatomical changes this stage would correspond to.

The *stage of latency* is complete, especially in the cases, in which the cerebral abscesses have persisted for decades. It is particularly in these cases, that one may speak of latency. For, if a patient frequently exhibits headaches, vomiting, convulsions, symptoms of cerebral pressure, or constant slight stupor, or focal symptoms—a state of affairs which by some, is designated as incomplete latency—it is a matter here of a slow progress, or a brief halt in the process, which can, at most, be distinguished from the terminal stage, but can not be contrasted with the stage of manifest symptoms. Of a real stage of latency, one may speak also in the cases already mentioned above, in which death suddenly appears in apparent health, and in which the autopsy usually reveals a large abscess, which had been situated in one of the "silent" portions of the brain (especially the frontal lobe) and which had then broken through.

The *symptoms of abscess of the brain in its active stage*—therefore that in which the diagnosis must be made—may be divided like those of tumor, into the *general and the local symptoms*. Among general symptoms we must distinguish those, which rest upon the presence of a purulent focus in the body, and, secondly those, which rest upon the influence of this purulent focus upon the brain as a whole.

Concerning the first group, it consists in reality only of a single symptom, *fever*. But it is very important, that in a very considerable number, possibly the majority of cases, there may be no fever at all, during almost the entire course of the disease, and that, therefore, the absence of fever should never be a reason against the assumption of a cerebral abscess. It is only with the rupture of the abscess, that fever always appears; but if one waits for that, it will be too late for effective treatment. In another group of cases, there is fever even in quite uncomplicated abscesses, usually, however, of only slight degree, and sometimes intermittent, but in other cases, high and continuous. On the other hand, subnormal temperatures are met with also.

In many cases, of course, it is difficult to decide whether the fever is due to the abscess or the fundamental disease, that is the concomitant changes (acute otitis, sinus thrombosis).

The *general cerebral symptoms* of abscesses are far less characteristic than those of the cerebral tumors. We traced nearly all the general symptoms of cerebral tumors to the creation of cerebral pressure by the tumor, and in so far as the abscess can cause spacial crowding, and can, by its internal tension, exert pressure upon the brain, it may, in exactly the same way as tumor, lead to cerebral pressure, and its phenomena, to headaches, choked disc, vomiting, stupor, slow pulse, etc. In many, possibly the majority of cases of uncomplicated brain abscesses, however, there is no pronounced cerebral pressure, as can be proven by measuring the spinal pressure by means of lumbar puncture, and by the absence of choked disc. But even in these cases there are often very grave general phenomena, especially a high degree of stupor and general epileptic convulsions, which then can be explained only by a poisoning of the brain through the bacterial metabolic products. On the other hand, even during the stage of manifest phenomena—therefore, we are not referring to the stage of latency—general phenomena may be absolutely lacking, and only local symptoms be present, as they are in every other local disease of the brain. If a meningitis is added to the abscess, we naturally shall have its general symptoms; we shall speak farther on about the differential diagnosis; here we shall only say, that a slight stiffness in the nape of the neck may also belong to the general phenomena of uncomplicated cerebral abscess, especially, but not exclusively so of cerebellar abscess.

The moment of *rupture* is frequently indicated by a very acute aggravation of the condition, occasionally by general convulsions, which are then very soon followed by the signs of meningitis. Sometimes, death occurs immediately with the rupture.

The *local symptoms of the abscess of the brain* are, per se, not characteristic. They have this in common with the local symptoms of tumor of the brain, that they appear comparatively often as irritation phenomena, especially also (corresponding to the seat of the abscess) as Jacksonian convulsions. On the other hand, neighborhood symptoms play a far smaller rôle in abscess than in tumor. There remains to be said that the local symptoms of abscess appear in an apoplectiform manner and may in spite of the progress of the abscess, retrogress again considerably. This seems to be the case especially in metastatic abscesses, where an apoplexy is simulated possibly by the disturbance in circulation, which occurs at the very moment of the embolisms

The outcome of abscess of the brain, unless it be opened by operation. is death. This is usually due to meningitis. But in other cases, death occurs, suddenly without the autopsy giving an adequate explanation; finally, the end appears with all the phenomena of death from tumor.

Special Diagnosis

The diagnosis of abscess of the brain is one of the most difficult and responsible tasks, with which the neurologist can be confronted. As is evident, from what has been said, regarding the ætiology, the anamnesis plays an important part, and the cases fall into two groups, so far as practical diagnosis is concerned: the first, in which the anamnesis points clearly to the possibility of a cerebral abscess, and the second, in which such an anamnesis or in fact any anamnesis can not be obtained, for the practitioner must always remember that in real life states of disease are not always as systematic, as text-books usually would have it.

Traumatism and *otitis* are the factors, which first and foremost indicate the possibility of an abscess.

Those traumatic cases are naturally very simple, in which the signs of a cerebral abscess develop in conjunction with a cranial injury, and in direct temporal sequence: the traumatic early abscesses.

Not quite so easy, but still readily recognized are the *traumatic late abscesses*, where the abscess is made manifest either without any injury to the osseous cranium at all, or after complete healing, whether prima or secunda intentione, has been effected. If a cranial traumatism is noted in the history of the patient, or a cutaneous or bony scar is discovered on the cranium, and if then, most frequently a few weeks or months after the traumatism, general or local cerebral symptoms appear, a cerebral abscess should always be thought of immediately.

In differential diagnosis, in all cases of traumatic origin, the epidural or subdural hæmatoma must be considered in the first place in contradistinction to abscess. The hæmatoma, however, usually comes on directly or almost directly after the accident, whereas the late abscess has a longer period of latency. It is true however that abscesses can not only develop in the course of a few days, but even within that time lead to severe phenomena and to death. The differentiation from meningitis may also have to be considered, especially in the rare cases, where there is a long free interval between the meningitis and the traumatism. Below, under the otitic diseases we shall return to the general distinction between meningitis and abscess.

In cases of existing *otitis*, especially in the chronic form, as soon as cerebral symptoms appear, the possibility of an abscess must always be considered at once. In these cases it is mostly a question of the differential diagnosis; *abscess-meningitis, or abscess-labyrinthine-suppuration.* The general definite symptoms of meningitis are described in another chapter. Should the diagnosis be in doubt, *lumbar puncture* will in every case be found to be an important aid in determining the presence of a meningitis, whether it be caused by rupture of the abscess, or by the otitis directly. Measurement of the pressure will enable us in these cases to make an approximately correct decision, if with a clear fluid the pressure should not be increased. This is a condition which, in case of doubt, may be supposed certainly never to be present in meningitis. On the other hand, a turbid discoloration of the lumbar fluid, which then is always accompanied by increase in pressure occurs only in *purulent meningitis.* Less definite conclusions are to be deduced from lumbar puncture when abscess is contrasted with *serous meningitis.* In it, the lumbar fluid exists under increased pressure, seems perfectly clear macroscopically; but, upon examination of the sediment, it is found to contain numerous lymphocytes. But this may also, though infrequently, occur in cases of abscess. Increased pressure without lymphocytes always speaks for abscess (or tumor) (cf. here what was said about lumbar puncture in tumor, p. 603 and 628).

It naturally speaks at once for meningitis if no local symptoms are present. Nevertheless, it is true that just those abscesses of the right temporal lobe occurring as a direct consequence of an otitis, and frequently also cerebellar abscesses, show only very slight or no local symptoms. In abscesses of the right temporal lobe, it is important to observe, as *Koerner* emphasizes, the neighborhood symptoms (which have already been described in the section on tumor of the temporal lobe), on the part of the oculo-motor, nerve ptosis, and difference in the pupils (p. 620).

On the other hand, in serous meningitis, especially if it is combined with non-purulent encephalitis, the frequency of which in otitis *Oppenheim* has emphasized (cf. the following chapter), there occur also focal phenomena, which may lead to the supposition of an abscess, where none exists. To give directions here for the individual case, is virtually impossible. Neither is the presence or absence of fever a great aid to diagnosis. In serous meningitis it is nearly always lacking, but, as has already been said, very frequently, also in abscess.

The differential diagnosis between *labyrinthine suppuration* and brain abscess, as, for instance, between the former and cerebellar abscess, may be very difficult, as the symptoms of a slight cerebellar disease may be simulated by a local meningitis and especially a slight encephalitis, more particularly as a definite determination of the origin of some symptoms common to both diseases, for instance, nystagmus, has not yet been made possible.

In otitic abscesses, differentiation from *sinus thrombosis* is also important. Its course, in contrast to that of abscess, usually is accompanied by high remittent fever and there are repeated chills. Pronounced local symptoms speak against sinus thrombosis; but the differential diagnosis is not always to be made, more particularly, since abscess and sinus thrombosis may occur simultaneously.

Especial difficulties attend the diagnosis of the endocranial complications of otitis in little children. Here one must always remember that general convulsions and conditions resembling meningitis may occur even in a pure acute otitis especially at its beginning.

As a last means of ascertaining the presence of an abscess in an existing otitis, operation upon the ear is to be performed, and the paths of the pus must then be traced; a means which may, however, also fail in its purpose.

Neisser-Pollack's cerebral puncture is of little value in cases of possible otitic origin.

Next to the otogenic abscesses, the *rhinogenic* are to be considered. Their location is the frontal lobe, and therefore they may run their course almost without local symptoms. They are very rare. From the standpoint of differential diagnosis, it is mostly cases of frontal headache, which raise the suspicion of cerebral abscess. In rejecting the diagnosis, cerebral abscess, because of the absence of the general symptoms—vomiting, stupor—one will have to depend upon, and find one's grounds in the fact that the absence of *every* local symptom is rare, even in disease of the frontal lobe. Differences in the reflexes must be looked for most carefully. If such are present (say the absence of the abdominal reflexes on one side, or the *Babinski*), the possibility of an abscess is naturally increased. In these cases, the local sensitiveness to pain, percussion and pressure has scarcely any value.

The abscesses, which do not arise from traumatism or purulent diseases of the cranial bones, are still more difficult to diagnose, and quite as difficult to recognize are those of traumatic origin, in which we have no anamnesis nor any traces of a traumatism. In all these cases, but especially in the cases of *metastatic* origin, all other local cerebral diseases are to be taken into consideration from a differential diagnostic standpoint. *Tumor* resembles abscess closest, since they both may lead to the same local and general phenomena. In these cases, a decision—if we exclude from consideration the cases of traumatic or otogenic origin—frequently can not be made upon existing conditions. The course in abscess, however, is usually far more rapid. Especially treacherous are those cases of abscess in which death suddenly occurs after several Jacksonian attacks. The absence of general symptoms speaks rather for abscess than for tumor, especially if one of the rarer possibilities of abscess causation is anamnestically determined as, particularly infectious diseases and purulent processes in the thoracic cavity. Especially when bronchiectases exist, is it quite easily possible to make a correct diagnosis of a metastatic cerebral abscess. If there is a history of an infectious disease, the differential diagnosis from *non-purulent encephalitis* may cause unsurmountable difficulties.

But even where it is to be differentiated from *softening* and from certain focal phenomena in *uræmia*, cerebral abscess may cause difficulties in all cases, where it is metastatic and where there are no motor irritation phenomena. Such, however, should always suggest an abscess, even though besides tumor (and pseudo-tumor p. 612) other, but very rare diseases, like *marantic sinus thrombosis* and *hemi-epileptic conditions* in senile cerebral atrophy are occasionally to be taken into consideration.

It has not yet been fully decided, whether and in what cases, if the presence of an abscess be suspected, *Neisser-Pollack's cerebral puncture* is justifiable. That surgeons, as a rule, are not inclined to attempt it in abscess, for fear of eventually infecting the meninges with the pus does not seem to be justified. For this danger exists just the same in cases where the abscess is laid open freely and under correct technique; and of course this puncture should be performed only in cases, in which, if pus be found, the operation can be performed at once. Therefore whenever an abscess is suspected, *Neisser's* puncture should be performed only in the surgical department of a hospital, and if the suspicion of an abscess is especially strong, no puncture should be made at all, but a free opening be made at once. Furthermore, it is probably not correct, that Neisser and Pollack consider the *negative result of cerebral puncture* as proof of the absence of a local disease. For it may happen, that even though various punctures are made into the deeper tissues, from the trephine opening above the abscess, still no pus be withdrawn, as the needle may always glide directly by the abscess. This is naturally even more possible in *Neisser's* puncture. But *Neisser's* puncture will certainly be of service in the cases in which a local diagnosis can not be made with sufficient certainty, especially in those, in which, as not infrequently happens, it vacillates between quite distant places, such as between frontal brain and cerebellum, and it might, furthermore, be advisable in a number of cases in which the diagnosis rests between hæmatoma and abscess.

The Local Diagnosis.—We will discuss the local diagnosis of brain abscess very briefly, since but very little can be mentioned that has not already been said in the general discussion on localization, or in the chapter devoted to cerebral tumors. Moreover, as a matter of fact, the local diagnosis of abscess presents far less difficulties in general than that of tumor. The general diagnosis of "abscess of the brain," is by far more difficult.

Abscess may occur in any part of the brain and the local symptoms are, in detail, quite the same as those in tumor; it is necessary to pay attention not only to the actual nervous irritation phenomena and symptoms of functional loss, but to the signs of local tenderness of the cranium to percussion and pressure as well.

Where we are dealing with an abscess of traumatic origin, as has been already remarked, it develops nearly always in the immediate vicinity of the traumatism.

In the otitic abscesses (with very few exceptions, as, for instance, when they are located in the stem) the differential diagnosis is concerned only with those in the temporal lobes, and with those of the cerebellum. Distinct, even though slight symptoms of low-grade aphasia, such as disturbances in finding words, and paraphasia are certain signs of left-sided temporal-lobe abscess. The right-sided temporal-lobe abscess can be recognized only by its neighborhood symptoms, i. e., oculomotor disturbances, hemianopsia, slight contralateral paresis.

The signs of cerebellar abscesses do not differ from those of cerebellar tumors. Should they be seated in the lateral parts of the cerebellum, their course may be absolutely devoid of symptoms, until rupture occurs and sudden death results from it.

In the otogenic abscess of the temporal lobe, as well as of the cerebellum, there can not be any doubt as to the side affected, excepting in cases where both ears are the seat of suppuration, since in the unilateral cases the abscess is always found on the side of the otitis.

Treatment

Prophylactic measures for the prevention of the formation of cerebral abscesses are, first, careful attention to cranial wounds, and, secondly, the thorough treatment of ear suppurations. There can be no doubt that by progress in these two directions, the frequency of cerebral abscesses has been considerably diminished. Traumatic late abscesses in city hospitals belong to the greatest rarities at present.

If a cerebral abscess is present, only *trephining* and free evacuation are of avail. The technical part of the operation is discussed by *F. Krause* in one of the last chapters of this book. If the suspicion of an abscess seems to the competent judge to be pressing, an operation must be decided upon at once, and carried out regardlessly. In particular, deep punctures will not suffice, but the brain substance must be laid widely open. The needle may, even in repeated punctures, miss the abscess, and a delay in the operation may cause death as a result of the rupture of the abscess. To make a diagnosis after the patient has died, and to bring the case to the autopsy table with the correct diagnosis, is no great feat in cerebral abscess. Of course, the diagnosis is sometimes so difficult, that, on the one hand, neither this case, nor, on the other hand, unfounded—at least not founded on the presence of an abscess—operations, where, for instance, a non-purulent encephalitis is found, are wholly avoidable. If, of course, a physician absolutely refuses to operate, or have operations performed, there is no possibility of unfounded operations. It is especially to be remarked that the metastatic cerebral abscesses also should be operated upon, unless it can be demonstrated that they are multiple; and in a large percentage of cases, they are not multiple. Demonstrable multiplicity, is, however, a contra-indication; almost the only one. A generally debilitated state of the patient is no contraindication; even comatose patients have been operated upon, and have recovered completely. An incipient purulent meningitis is not regarded at the present time, everywhere, as an invariable contraindication, though the chances, if the lumbar puncture brings forth distinctly turbid fluid, are very small.

Although operation offers the only possibility, it by no means gives an assurance of recovery, even if performed at the right time, and complications like meningitis, sinus thrombosis, and pyæmia do not occur. The reason for the failure, apart from the dangers of the operation, may be that only part of the abscess was evacuated, or that a second abscess is present, or that a progressive softening is spreading from the region of the abscess. The percentage of cases of cerebral abscess cured by operation varies so extraordinarily, even in the statistics of prominent operators, between 25 and 95%, that here probably, the material, or the choice of it for operation, must have been the determining factor. According to general opinion, the cerebellar abscesses present particularly poor chances for operative cure.

5. NON-PURULENT ENCEPHALITIS

BY

M. LEWANDOWSKY (Berlin)

Probably no other cerebral disease is so undefined and variable in its foundation and origin, as non-purulent encephalitis. We meet with the remarkable fact, that under the name encephalitis, inflammation of the brain, a number of diseases are described, the inflammatory nature of which is not only doubtful, but even disproven in an anatomical sense, and that other diseases, the inflammatory nature of which is determined with certainty, are by convention and for practical reasons not included in a discussion of encephalitis. To the latter belong, besides the purulent inflammation, the brain abscess, also progressive paralysis of the insane, and apparently also the trypanosomatic diseases, among them the sleeping sickness.

Particularly because of the multiplicity of the clinical pictures, pathological anatomy should be of great service in producing a disease entity. But our knowledge of histology and histopathology of the brain, was, up to comparatively recent times, in a far from consolidated condition, and even the present state of our knowledge has not been employed sufficiently by all investigators nor yet in a sufficient number of clinically observed cases.

Thus even in regard to pathological anatomy, encephalitis has remained to the present day a general term; the reason for this lies partly in the fact that the definition of the inflammation concept has its own peculiar difficulties, when applied to the brain, since we are not concerned here, as in other organs, with a single parenchyma and an apparatus of vascular connective tissue, but in addition to the functioning components of the nervous tissue, there is to be considered also a supporting tissue of ectodermal origin, the glia. If since *Cohnheim's* investigations most authorities agree, that inflammation is characterized by the migration of white corpuscles and the effusion of fluid through the non-ruptured vascular wall, this must certainly hold good for encephalitis also. At any rate, if this were not so, all forms of focal disorder, which are accompanied by the egress of white corpuscles would otherwise have to be considered as an entirely different kind of inflammation. If one looks through the literature from this point of view, very little that is satisfactory will be found. For one group of authors includes all reactive processes of the cerebral tissue under the term encephalitis; for instance, the results of an aseptic puncture in the cortex of the brain. If this is done, there are very few affections of the brain which may not be thus included under encephalitis, for *Nissl* demonstrated that changes of this kind appear everywhere, that wherever cerebral substance is destroyed, or wherever necrotic cerebral substance or a foreign body is surrounded by living tissue, the latter reacts in a perfectly definite and always in the same way. It makes no difference what type of injury caused the destruction, whether it was a stab, a hemorrhage, or a cauterization, the same reactive processes always follow; at first they become manifest mainly in the connective tissue about the blood-vessels (mesodermal type of *Schroeder*) and reparative phenomena on the part of the glia show themselves at the beginning, only in the periphery of this mesodermal reaction area, later and very gradually these changes advance into the realm of the connective tissue, replacing it entirely or in part. It is therefore a matter of purely reparative processes, and, as a matter of fact, of such processes in which migration of white corpuscles through the walls of the vessels plays no part at all. If this would be called inflammation, it would be necessary to say that every hemorrhage in the cerebral substance leads to an inflammation, and also to call the formation of every scar in other organs an inflammation. The general broadening of the conception of inflammation has been in large part caused by the view, that the presence of the so-called *granular cells* is virtually to be regarded as proof of an inflammation, because they originate from the white cells in the blood, and clinicians therefore have been inclined to diagnose inflammation, wherever the granular cells were found. It was left to Nissl and his school, to bring forth the proof that the fatty granular cells do not originate from white corpuscles, but in part from fixed connective-tissue elements and cells of the blood-vessel walls, the other from glia cells; and that it is altogether doubtful whether leucocytes in the nervous system are transformed into granular cells in any great numbers. The fatty granular cells are therefore not a sign of inflammation. To inflammation proper, in the pathologic-anatomical sense, as has already been stated (besides abscess), belong progressive paralysis and also as it seems the trypanosomatic diseases, especially sleeping sickness. But these are not treated of in the chapter on encephalitis. What still remains for encephalitis in the strict pathologic-anatomical sense, one hardly knows in the face of *Schroeder's* explanation, that in all the cases handed over to him by clinicians and anatomists as "hemorrhagic encephalitis," nothing else could be found histologically but hemorrhages (in the sheaths of the vessels or in the tissues), never genuine inflammatory processes, and that in most cases it was a matter of embolic and thrombotic processes. For one definite form, as we shall soon see, this beginning with hæmorrhages and the absence of actual inflammatory processes has been firmly established. It is probably a consolation in this condition of things, that the demarcation of the concept of inflammation is almost as much disputed in the pathology of other organs. We shall therefore in the following have to proceed mostly from clinical points of view.

I. POLIENCEPHALITIS HÆMORRHAGICA SUPERIOR (Wernicke)

Etiology.—Poliencephalitis hæmorrhagica superior is a *disease of habitual drinkers*, especially of the drinkers of spirits. *Wernicke*, who established the disease picture, had among three cases, two drunkards and a young girl, whose disease, classified under this head by Wernicke, came on four weeks after her recovery from poisoning by sulphuric acid. Though later the diagnosis of this last case as poliencephalitis hæmorrhagica, was disputed, it is nevertheless possible, that analogous pictures occasionally originate through other toxic influences than alcohol, especially through meat and sausage poisoning. Ætiologically, clinically, and even in regard to pathologic anatomy, however, this encephalitis of habitual drunkards exhibits so consistent a uniformity, that a special place must be given to it.

Pathologic Anatomy.—In spite of its name, encephalitis hæmorrhagica superior is an example of a *non*-inflammatory disease in the above-described anatomic sense. Macroscopically the findings are seen to consist of characteristic small *hemorrhages* in the vicinity of the third and fourth ventricles, as well as of the Sylvian aqueduct. They are usually only as large as a millet or a pin head; occasionally, however, the hemorrhages are larger; frequently they are to be discovered only by microscopic examination. The microscopic examination (*Bonhoeffer*, *Schroeder*), shows nothing but small hemorrhages into the vessel sheaths or into the tissue, which induce the characteristic reaction on the part of the vascular connective tissue, that common reaction (cf. above) which is often mistakenly regarded as inflammation.

Symptomatology.—It is usually chronic drinkers of spirits, who have passed through one or more attacks of delirium tremens, and in whom the anamnesis gives the usual indications of chronic alcoholism more or less completely, who are attacked by the disease. A few days or weeks before the actual outbreak, prodromal symptoms may appear in the form of vertigo, headaches, and frequent vomiting. Usually the disease begins like delumiri tremens, and only a few days later the symptoms of acute encephalitis develop. To the delirium there are soon added severe stupor and great weakness. The characteristic paralyses of the eyes develop, and the patient usually dies in the course of a few days or weeks. In a considerable number of cases, however, to which we shall return later, the disease does not end fatally. The temperature is frequently subnormal, and but rarely rises to 100–101 degrees. The pulse is nearly always accelerated. The disease may appear complicated by polyneuritic symptoms. Neuritis optica with hemorrhages has been repeatedly observed. The gait and speech correspond, on the whole, to those seen in simple severe delirium; there are present severe ataxia, sometimes cerebellar in type, tremor of the tongue and hands; speech is stumbling, sometimes incoherent. In some cases, hemipareses and even transitory spastic conditions have been observed. A slight stiffness in the nape of the neck is not unusual. The psychic condition is that of alcoholic delirium; there are anxious confusion, hallucinations, disorientation, etc., until the stupor blots out these traits. The disease may begin with or be interrupted by epileptic attacks.

The cases that do not end fatally, may become transformed into a condition of chronic delirium, especially into Korsakow's psychosis; in this, however, the signs of paralysis of the muscles of the eye may disappear.

The *paralysis of the muscles of the eye* frequently appears in the associated form, but other forms occur also; for instance, partial ophthalmoplegia on both sides, or bilateral abducens paralysis. Wernicke traced these paralyses of the muscles of the eye back to the hemorrhages in the ventricular gray matter near the nuclei of the upper cerebral nerves, and thus the entire disease received its name. The inner muscles of the eye are not, as a rule, affected.

But there is no doubt that, as especially *Bonhoeffer* explained, these very symptoms of poliencephalitis superior form only a part of a severe general disease of the brain, especially of the cortex of the cerebrum, which develops on a foundation of chronic alcoholism, just as in the various forms of alcoholic delirium, paralyses of the eye muscles frequently appear. It would seem very probable that the poison, which develops under the influence of chronic alcoholism, and which causes the poisoning of the entire nervous system, increases also the permeability of the vessels, so that hemorrhages occur. It has been demonstrated that this is the case not only in the cerebral vessels, but also in those of other parts of the body. Why in the cases of encephalitis hæmorrhagica superior the vessels of the quadrigeminal region react so energetically, is like all such regional affinities, obscure.

The **prognosis** is evident from what has been said. The danger of a fatal termination is very great. In the beginning, no prognosis of any individual case can be given. If death does not occur, the sequel is usually a slow, long lasting Korsakow's psychosis, from which the patient rarely recovers completely.

II. THE MAIN GROUP OF ENCEPHALITIS

This second group is by no means uniform. It is possible that the cases brought together here, will be annexed at some future time to those of group

I on the one hand, to those of group III on the other; both are better differentiated; at the present time, however, this is not possible.

Etiology.—The ætiological factors are, first of all, of a toxic-infectious nature, and the type of this group is the *influenza encephalitis*, our knowledge of which we owe to *Leichtenstern* especially. The influenza bacillus itself, was first isolated by *Nauwerck* in the central foci and in the contents of the ventricles, although the clinical connection had been recognized some time prior to this. It is not even certain that encephalitis during and following influenza must always be of bacterial origin; it is possible the bacterial metabolic products suffice; it is said that chlorosis presents a peculiar predisposition for the development of encephalitis.

Epidemic cerebro-spinal meningitis may also lead to the development of an encephalitis (*Klebs*, *Leichtenstern*), and especially in cases in which no infection of the cerebral membranes is made evident. Its exciting cause, *Weichselbaum's* diplococcus has been demonstrated in the foci. Occasionally, after the corresponding infectious diseases, pneumococci, typhoid bacilli and even the bacilli of anthrax have been found in the cerebral foci, and must therefore be regarded as possible excitants of encephalitis. But probably in a number of cases not the micro-organisms themselves, but only their toxines are concerned. In other infectious diseases in whose train encephalitis may appear, it is uncertain, in some at least, because of our insufficient knowledge of the exciting cause of the infection, whether strictly speaking, an infectious or only a toxic cause is concerned. We must mention, in addition to those above, measles and scarlet fever, but there is probably no infectious disease in the course of which encephalitis has not been observed.

Encephalitis appears especially frequent in purulent ear diseases, usually associated with serous meningitis.

Encephalitis has further been observed after poisoning by meat, sausage, fish, fungi and sulphuric acid. The nosological and pathologic-anatomical character of these cases has not been definitely determined. On the other hand, poisoning by carbonic oxide seems to be able to produce exactly the same picture as the ætiological factors of encephalitis.

Encephalitis has appeared, furthermore, as a *primary disease* without any known antecedent infection. Here belongs, first of all, the encephalitis of children. That a large number of cerebral infantile paralyses are to be traced back to an encephalitis (in the broad sense of the word)—the only one that is tenable—seems unquestionable, considering the theories of *Marie and Strümpell.* Whether one uniform infectious agent is responsible for these kinds of encephalitis, as it is assumed for the majority of cases of poliomyelitis, though it has as yet not been isolated, is doubtful (cf. also below under group III). It is also doubtful whether tuberculosis can cause an ordinary hemorrhagic encephalitis. On the other hand typical large foci are occasionally observed in tubercular meningitis.

There is no doubt that *traumatism* has an unquestionable influence on the production of changes, which we, for the present, call encephalitic. Of course, as has been stated, we never consider the direct consequence of an aseptic injury, for instance, a contusion of the brain, as an encephalitis; but there are cases, where, with a latency of days or weeks after a traumatism, which has not caused any external injury to the cranium, there develops an encephalitis or a focalized hemorrhagic disease. It has been asserted that in such cases an infection is the exciting factor, but that is probably not true for all cases, particularly as fever may be absent throughout.

Pathological Anatomy.—As regards the pathological anatomy, we may point to what was said in the introduction to this chapter. It is to be added, that the so-called encephalitis, even apart from encephalitis hæmorrhagica superior, in most instances is a hemorrhagic condition; the hemorrhages occur either as a large circumscribed focus, or in a well circumscribed space a number of small, sometimes microscopic, foci are found close to each other, or the hemorrhages are quite disseminated. It has not yet been cleared up, whether these hemorrhages are secondary or, as in *Wernicke's* form, primary. In several cases thrombosis of the small vessels is reported; this may possibly have been secondary, but could as readily have been primary. It is also to be remembered that encephalitis is rather frequently observed in combination with sinus thrombosis. The hemorrhage may be so severe that the picture is scarcely to be distinguished from that of a cerebral hemorrhage. Whether *hemorrhagic* encephalitis, as such, is to be considered as a different disease from the *non-hemorrhagic* variety, is uncertain, but not probable.

The histology of the *non-hemorrhagic* forms has not yet been definitely established. In fact in some of the cases nothing at all has been found macroscopically. Microscopically there are a number of reports. From the point of view of Nissl's investigation, the field has not yet been covered. Especially obscure is the position of disseminated encephalomyelitis (poliencephalomyelitis), in which the supposedly inflammatory changes are scattered over the entire nervous system. Here differentiation from *multiple sclerosis* is the most important point. As a matter of fact, in quite acute cases of this kind, even in recent foci, only the banal changes of multiple sclerosis have been found.

Synchronous with encephalitis, there are frequently found changes in the cerebral membranes.

The outcome of encephalitis, especially of the hemorrhagic variety, is the formation of scars and cysts. It is not known whether, in a number of cases which have ended in clinical recovery, *complete* anatomic restitution has occurred.

Symptomatology.—A very large number of cases of encephalitis of this group are characterized by an *acute course.* In the great majority there is fever; it may be either continuous or remittent. In infantile encephalitis the fever may precede the focal disease—in that of adults this does not, as a rule, seem to be the case. The type of this disease is the influenza encephalitis. It usually breaks out again with renewed fever, after the real influenza has run its course, sometimes after full convalescence, sometimes after general prodromal symptoms have preceded for days. Somnolence and deep stupor supervene very quickly. Vomiting may be present even at the beginning. Psychic conditions of excitement are rare in the initial stage. The entire cranium may be tender to percussion or only in circumscribed spots. There is slight stiffness at the nape of the neck. General convulsions are the rule in the encephalitis of children; also they occur in full grown adolescents, but less frequently. There are cases, in which even in the adult after the presence of fever for several days, general convulsions appear suddenly, become more and more frequent and the disease without any other symptoms than these, ends fatally very quickly.

Optic neuritis occurs occasionally. Focal phenomena may be present from the first, or appear only during the course of the disease. We shall not attempt to describe them individually. Since encephalitis may attack any part of the brain, all sorts of localized phenomena of functional loss may appear. Hemiplegias, aphasias, cerebellar and pontine symptoms, paralyses of the cerebral nerves due to affection of the nuclei, sometimes alternating paralyses, etc. Naturally the same focal symptoms as in *Wernicke's* form may be present. If the focal phenomena are not very severe, they will be discovered only with difficulty, because of the general stupor.

In a minority of cases, which nevertheless seem to belong to the infectious-toxic group, the development may be somewhat different. We have already said that fever may be absent. Consciousness also may remain comparatively intact. The focal phenomena may develop very gradually.

In many cases the course of encephalitis is acute or peracute, leading to death in a few days or weeks. Cases in which the disease is subacute, with stages of exacerbation but in which the final fatal end can not be prevented, are less common. Recoveries with defects corresponding to the extent of the destroyed area are specially frequent in children, but occur also in adults. Some of the permanent sequelæ in children are described in the chapter on cerebral infantile paralysis. In a considerable number of cases there is, however, total recovery and moreover even in those, that have begun with severe general and focal symptoms.

III. ENCEPHALITIS AS A PARTIAL PHENOMENON OF THE HEINE-MEDIN DISEASE

Etiology.—By the *Heine-Medin* disease we understand with *Wickmann*, an entire group of disease pictures, which are connected by a common ætiology, the underlying factor of which, as is proven by epidemiological investigations, must be a uniform virus, though its nature has not as yet been discovered. The prototype and the most frequent of these disease pictures is the poliomyelitis spinalis anterior, but there can be no doubt, that the same process may be manifested in the brain also. Investigators have gone so far as to trace the encephalitis and the poliomyelitis of childhood in general to the *same* unknown virus. But the fact, that, in large epidemics of poliomyelitis, only isolated typical hemiplegias, or cerebral infantile paralyses have been observed must be quoted against this. The hemispheres, which are the main seat of the encephalitis in children, are apparently but very rarely attacked by the virus of *Heine-Medin's* disease, so that the large mass of infantile encephalites must be classed with the preceding group. Far more common in this group is an affection of the brain stem, the bulbar and the pontine form. Cerebellar affections occur also.

Pathological Anatomy.—*Heine-Medin's* disease seems also in its pathologic histological picture to form the type of the genuine inflammation with leucocyte infiltration of the sheaths of the vessels. Hemorrhages occur only in exceptional cases. The nerve cells may also be directly attacked by the active virus, so that they may be destroyed quickly. Under such circumstances an absorption by leucocytes (neuronophagia) has been observed.

The course of the disease is quite different from that of the first named forms. The disease like the first group presents a febrile character, but while in the former the fever frequently does not appear until the height of the disease is reached, it is, in this, probably always the first symptom; it may be of short duration, so that only after an apparent recovery the consequences of the disease are noticed. In the initial stage, headache and vomiting, also slight stiffness in the back of the neck are common. The disease preferably attacks children. The consciousness is never so severely impaired as in the first two groups, scarcely more than corresponds to the degree of fever. Even though no epidemics exist, the recognition of the cases will cause no difficulties on account of these differences.

The localization of the disease shows itself in the bulbar and pontine form analogous to poliomyelitis by a paralysis of the cerebral nerves; accordingly as the medulla oblongata with the hypolgossal, facial and abducens, or the quadrigeminal region with the oculomotor is more severely affected one may speak of a poliencephalitis inferior or superior, but the latter must not be confused with Wernicke's form. The corresponding paralyses of the cerebral nerves are the main symptoms, and are almost never symmetrical but almost always unilateral. As in the spinal cord, the disease is not entirely restricted to the motor cells, but may affect also the neighboring tracts, the motor and sensory, so that corresponding disturbances, which, however, are never very severe, may be observed. The cerebellum may in rare cases be affected alone, and the disease then presents nothing but the phenomena of cerebellar ataxia. If the disease attacks the cerebrum proper, which, as we have said, is rare in this group, the signs are the same as in the other forms. Neuritis optica is very rare.

The course threatens life only in a minority of cases, especially (if we except the cases of ascending and descending paralysis which will be discussed immediately) only in the very rare cases in which, because of a bilateral paralysis of the cerebral nerves, swallowing is impeded, and thus assistance afforded to the development of complications. In a number of cases, even severe paralyses disappear completely. In the majority, more or less pronounced pareses of the musculature of the head and face remain.

A special variety of *Heine-Medin's* disease may be formed by an *ascending paralysis of Landry's type.* Wickmann has observed this in epidemics and the histological findings in non-epidemic cases confirm it. Here, then, the disease attacks the brain stem ascendingly from the spinal cord. This form is very unfavorable prognostically, and nearly always leads to death in a short time (by paralysis of respiration and deglutition). But not all forms of ascending paralysis are poliomyelitic and polioencephalitic; we shall return to this topic immediately.

IV. ENCEPHALITIC SYMPTOMS WITHOUT ANATOMIC FINDINGS

A number of cases, which ran their course under the picture of an encephalitis, came to autopsy at the very height of the disease, and yet it was impossible to discover any pathological findings. In these cases a pure poisoning of the nervous system, or certain of its parts must be assumed. The appearance of acute bulbar paralysis, or of poliencephalitis superior et inferior is very frequent in cases of poisoning by sausage, meat, fish, and fungi, as also in other cases of obscure ætiology. The important symptoms, here, are the paralyses of the cerebral nerves. But even cases, which exactly resemble encephalitis hæmorrhagica, because of the presence of fever, deep stupor and focal phenomena on the part of the cerebrum (aphasia, hemiplegia, pseudo-bulbar paralysis) are observed again and again without any pathological findings, and preclude the possibility of differential diagnosis from encephalitis.

Here we are not even referring to the focal phenomena without corresponding findings in uræmia, or chronic nephritis, though these may be related to the cases in question, but the cases of entirely unknown ætiology.

Finally we must say that ascending paralysis with involvement of the cerebral nerves, may appear without any pathologic-anatomical findings (genuine *Landry's* paralysis), hence it can not be clinically differentiated from the cases of Heine-Medin's disease portrayed in the previous group.

Differential Diagnosis of Encephalitis.—There is a certain amount of difficulty in defining a disease so uncertain in many directions as is that of encephalitis. First of all, all forms of encephalitis may make a meningitic impression, since, as a matter of fact, the meninges may be involved in the disease. Lumbar puncture at least sometimes affords a means of differentiation. It scarcely ever reveals a perceptibly increased pressure. In most cases, the cerebro-spinal fluid is perfectly clear, but may also be slightly hemorrhagic; it may also contain leucocytes. Greater turbidity points to meningitis. For tubercular meningitis, there is always the possible finding of tubercle bacilli.

Concerning that form then, which runs its course with large, single foci, confusion is possible with *foci of softening*, with *hemorrhages* and with *abscess*. Fever which is usually present in encephalitis serves to distinguish the disease from thrombotic softening and hemorrhage. Besides the age preferred by encephalitis is that of youth, while thrombosis and hemorrhage attack old arteriosclerotic individuals. As contrasted with softenings, based on syphilitic vascular diseases, the presence of specific syphilitic phenomena (reflex pupillary rigidity) or other signs of syphilis will be of importance for the decision. But we must note that extensive encephalomalacias without involvement of the larger vessels have been described; they might justly be classified as encephalitis. Even at the autopsy the differential diagnosis between hemorrhage and encephalitis in young persons may cause difficulties, since even in cases of encephalitis hemorrhagic cysts, etc., may develop. This must be considered if a question arises after a traumatism, whether a late apoplexy or encephalitis is present. According to some authorities, late apoplexy is always an encephalitis hæmorrhagica.

Of greatest practical importance is the differential diagnosis between encephalitis and *abscess of the brain*. This was discussed in the chapter devoted to the last named disease. This differential diagnosis is sometimes difficult, symptomatically even impossible, so that when the localization is ascertained, Neisser's puncture must be employed to decide it.

Concerning the differential diagnosis of the forms accompanied by paralysis of the muscles of the eye, that of *Wernicke's* form could scarcely be missed, and no more so that of group III (*Heine-Medin's* disease). The rest, namely those forms founded neither upon alcoholic basis, nor upon infection with the virus of *Heine-Medin's* disease, may cause confusion with *polyneuritis*. This, however, is not so dangerous since no practical consequences follow the confusion and in many cases *polyneuritis* and polioencephalitis occur together.

In contradistinction from the disseminated forms of encephalitis, multiple sclerosis has also to be considered, which however by some is brought into genetic connection with polioencephalomyelitis. Still it seems to be very probable, that it is a matter of two absolutely different processes. For even the most acute multiple scleroses seem to show a different histological picture from encephalitis, namely from the first glia proliferation (with new formations in vessels). Besides the axis cylinders in multiple sclerosis nearly always remain intact. With a very acute beginning in multiple sclerosis, which also, as in encephalitis, even though only in very rare cases, may be ushered in with severe stupor, diagnosis may be difficult for a time. Fever will always be absent in multiple sclerosis. It will be very rare, after all, to mistake a multiple sclerosis for a polioencephalitis. But probably a relatively large number of cases get into the literature, as cases of poliomyeloencephalitis, which are probably nothing else but multiple sclerosis with an acute course.

Treatment.—It has not yet been proven that any therapeutic measures can influence the anatomic process of encephalitis in any of its forms. A specific or a specifically effective treatment has not as yet been found. In the forms due to an excitant, as especially the poliencephalitis hæmorrhagica superior of habitual drunkards, the procedure naturally must be according to the general rules for the treatment of delirium; narcotics should be given, though naturally as little as possible (veronal, chloral, with very severe excitement hyoscin). In not too great excitement and with satisfactory heart action, one may, where it is possible, advantageously use continuous baths. For the rest, supervision and excitation of the heart's action is the main task, for which naturally we can not give directions in this place. Besides the above mentioned treatment, in cases of acute hemorrhagic encephalitis, according to the need of the individual case, leeches have been employed upon the back of the neck or in the region of the temples, possibly with some success. Not only in the cases, in which an intoxication from the bowels is suspected, but in all cases, thorough evacuation of the bowels must be attended to, for which calomel is best suited.

If, after an encephalitis has run its course, irritation phenomena remain, especially in the form of Jacksonian convulsions or general epilepsy, from which we must conclude that definite scars or cysts are present, the operative opening of the skull and evacuation of the cysts is to be considered, in certain conditions also the extirpation of the pathologically altered cortical centers (cf. *F. Krause*, Chapter XII). These cases almost exclusively occur as resulting phenomena of *infantile* encephalitis.

6. DISTURBANCES IN THE CIRCULATION IN THE BRAIN

(Anæmia, hyperæmia, laceration of the vessels, occlusion of the vessels, syphilitic vascular changes, general arteriosclerosis, meningeal hemorrhages and hæmatoma of the dura mater, sinus-thrombosis.)

BY

C. HIRSCH (Göttingen)

I. ANATOMY[1] AND PHYSIOLOGY OF THE VESSELS OF THE BRAIN

The arteries of the brain arise from the *carotid* and *vertebral.*

The cerebral branches of the *carotis interna* branch out on the surface of the pia mater (vascular membrane). In this membrane arise the arteries that enter the brain.

These cerebral branches are:

(a) The a. *cerebri anterior.*
(b) The a. *cerebri media* (art. fossæ Sylvii) with the AA. lenticulo-striatæ.
(c) The a. *chorioidea.*
(d) The a. *communicans posterior.*

The a. *cerebri anterior* courses medially and forwards behind the origin of the tractus olfactorius, it crosses the optic nerve, and runs to the median longitudinal fissure of the cerebrum. Hence it courses forwards and upwards to the genu and to the upper surface of the corpus callosum. At the splenium corporis callosi it bends downwards. It sends off branches to the corpus striatum, to the under surface of the frontal lobe and to the upper surface of the hemispheres. In front of the chiasma at its entrance between the frontal lobes of both hemispheres both arteries are connected by the short transverse a. communicans ant.

The *a. fossæ Sylvii* (a. *cerebri media*) is the largest branch, sending many branches backwards into the Sylvian fossa. Near its beginning, it sends small branches through the substantia perforans anterior to the corpus striatum. From it branches also go to the frontal lobe, to the insula and to the temporal lobe.

The *a. chorioidea* may also be considered as a branch of the a. fossæ *Sylvii.* It courses under the gyrus uncinatus to the plexus of vessels in the inferior cornu of the lateral ventricle.

The a. *communicans posterior* arises near the trunk end of the carotis and courses over the cerebral peduncles to the *a. profunda.* No branches go out from it. It closes the circle of arterial trunks at the base of the brain—

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[1] According to *C. Gegenbaur* and *F. Merkel,F . Merkel's* handbook of topographical anatomy gives the practitioner the most definite anatomic foundations for his clinical work, Cf. also *Spalteholz,* hand atlas of human anatomy. *Corning*, text-book of topographical anatomy.

circulus arteriosus Willisii; its completion posteriorly is formed by both aa. profundi cerebri and anteriorly by the a. communicans anterior (Fig. 177).

The *a. vertebralis* represents the most important branch of the subclavia. Both a. vertebrales are united behind the pons into the a. *basilaris*.

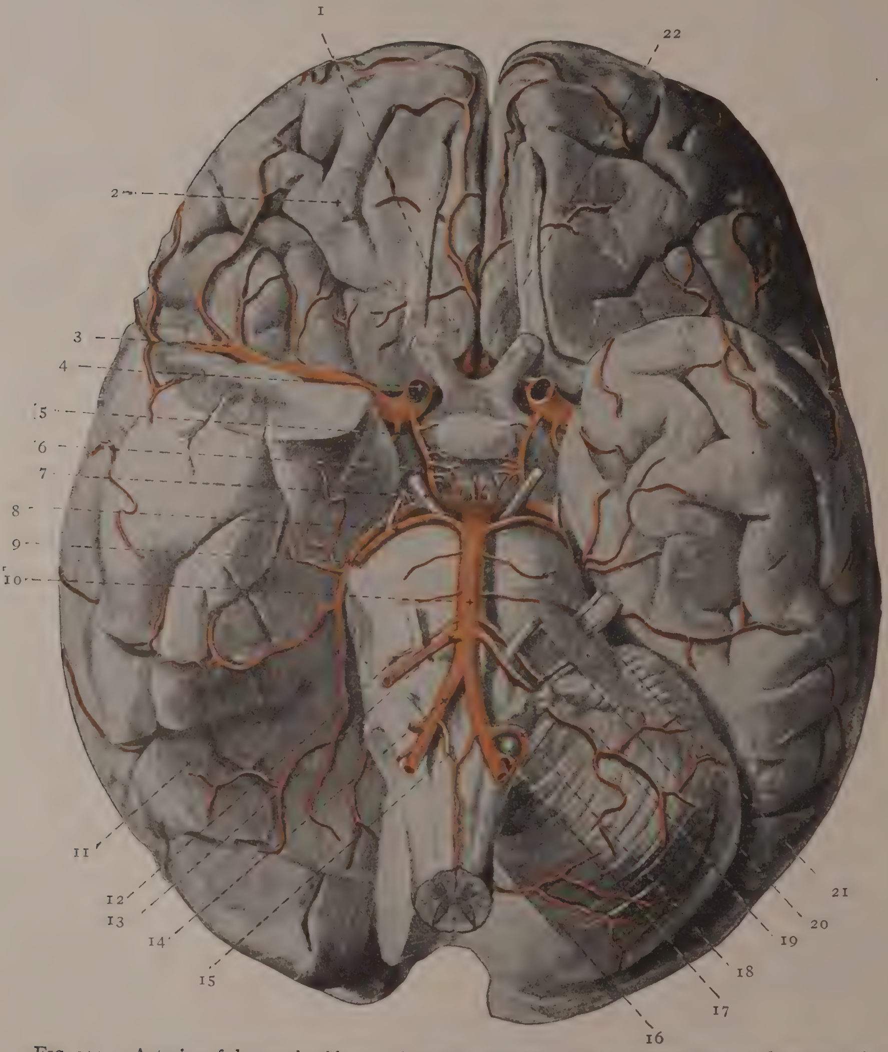

FIG. 177.—Arteries of the cerebral base. (*From Spalteholz.*) The vertex of the right parietal lobe and the right side of the cerebellum are removed. 1, A. cerebri ant. 2, Lobus frontalis; 3, A. cerebri media; 4, A. carotis int.; 5, A. chorioidea; 6, A. communicans post.; 7, N. occulomotorius; 8, A. cerebelli sup.; 9, A. cerebri post.; 10, A. basilaris; 11, Lobus occipitalis; 12, A. auditiva interna; 13, A. cerebelli sup. ant.; 14, A. vertebralis; 15, A. spinalis ant.; 16, Cerebellum; 17, A. spinalis post.; 18, A. cerebelli inf. post.; 19, N. facialis; 20, N. abducens; 21, Ramus ad. pontum; 22, A. communicans ant.

The peculiar course of the artery at the first and second cervical vertebræ appears to be purposely fitted to the mechanism of the movement of the head (*C. Gegenbaur*).

Upon rotation of the head the artery of one side is markedly compressed (that one that is turned from the face), while in that on the other side circulation is made easier. By this compensatory behavior, the passage of blood to the a. basilaris is regularly preserved (*Gegenbaur, L. Gerlach*).

The *a. basilaris* divides into the aa. profundi (a. cerebri post). Before doing so it sends off the following branches:

(a) one to the pons Varolii
(b) the a. auditiva interna
(c) the a. cerebelli anterior inferior
(d) the a. cerebelli superior.

The *a. cerebri posterior* (*a. profunda cerebri*) courses laterally in front of the pons; here it embraces the n. oculomotorius. It unites with the a. communicans posterior, passes around the cerebral peduncles upward to the under surface of the temporal and occipital lobes of the cerebrum, where it breaks up into branches.

At its origin fine branches begin, which course between the cerebral peduncles to the third ventricle. They pass through the *substantia perforata posterior*.

From the upper division of the *a. profunda cerebri*, branches go to the *quadrigeminal bodies* and the tela chorioidea.

From these three large cerebral arteries (a. cerebri anterior, media, posterior) and the circulus arteriosus, branches spring during their basal course, which penetrate directly the substance of the brain and pass to the cerebral ganglia and the neighboring medullary substance. These branches are said to show no anastomotic connections.

In the rest of the surface of the brain, the arteries form the so-called pial vascular network. The neighboring vessels anastomose here and hence shorter or longer little twigs pass into the cortex and the subcortical medullary substance. A *central* and a *cortical arterial network* (basal region, cortical region of *Heubner*), therefore, is distinguished.

Merkel is correct in emphasizing, that with so clear an anatomic separation between the vascular regions, the diseases of the ganglia and of the cortex of the cerebrum are to be distinguished sharply also, at least in so far as the cause of the disease is to be sought in lesions and occlusions of the vessels.

The statement of *Heubner and Duret*, that all the small arteries of the basal region are endarteries, i.e., form no anastomoses, needs, in my opinion, to be subjected to reexamination, since the conception of the endartery has had to be abandoned even for the coronary vessels of the heart (*W. Spalteholz and C. Hirsch*).

If *places of predilection for emboli to lodge* are looked for in the course of the individual arteries it will not be difficult to find them.

Since the *a. cerebri media* (*Sylvii*) represents the real continuation of the main trunk of the carotis, it is not surprising, that an embolus, that comes from the carotis, must reach just this very artery and not the a. cerebri anterior, which leaves the carotis almost at a right angle. (*Duret.*)

According to *Heubner* the area of origins in the anterior part of the basal region must be divided into three parts: 1. the *a. cerebri anterior* from its beginning to the *ramus communis anterior*, 2. the *first centimeter* of the course of the a. cerebri media, and 3. the *second centimeter* of the same artery (Fig. 178).

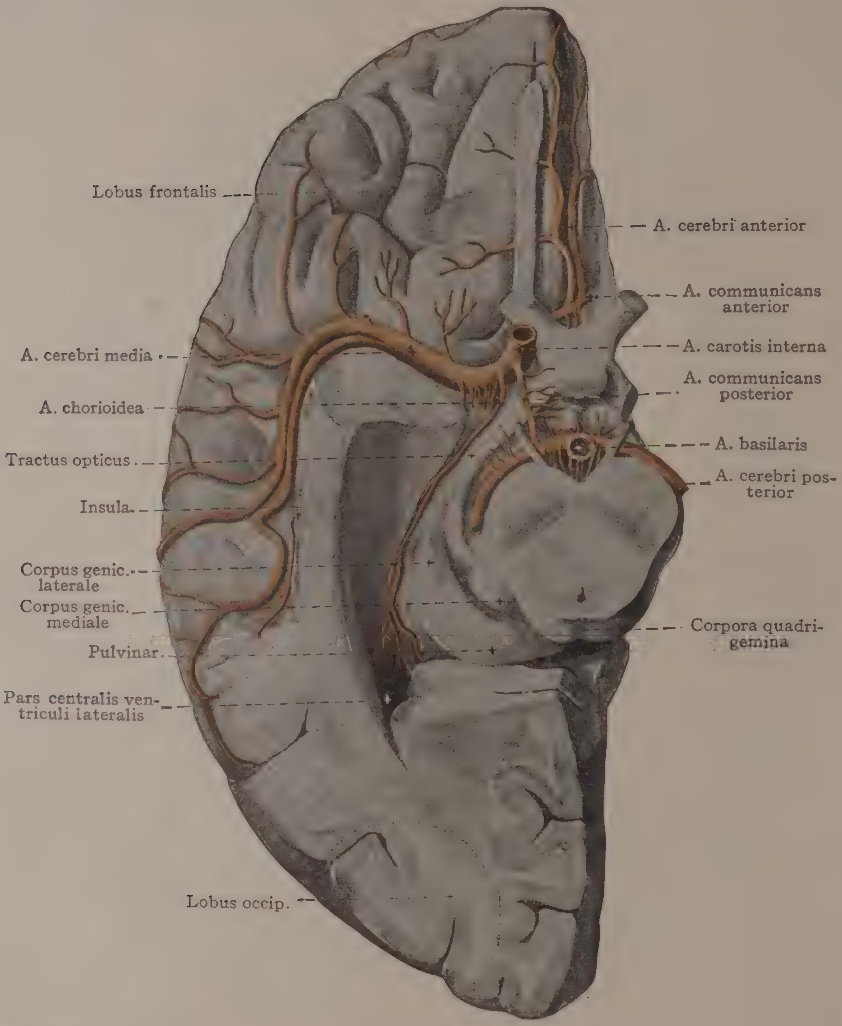

FIG. 178.—A. cerebri media and A. chorioidea. (*From Spalteholz*). The parietal and occipital lobes are removed to the opening of the cornum inferius ventriculi lateralis, to expose the fossa cerebri lateralis (Sylvii); the cerebellum and the pons are cut off immediately before the anterior, upper edge of the latter.

From the third part of its course, little vessels branch off, which reach the brain through the substantia perforata lateralis. According to *Heubner*, they supply the lateral and upper parts of the lenticular nucleus, the middle part of the caudate nucleus and the external capsule. *Duret* points especially to one of these arteries, which passes by the transition from the lenticular nucleus to the internal capsule, then divides into several branches, which end in the lenticular and caudate nuclei. *It is a point of predilection for the hemorrhages observed here.*[1] (A. cerebri media with the aa. lenticulo-striatæ which spring from it, *Monakow*.)

From the a. cerebri posterior, a few branches pass through the substantia perforata media to the quadrigeminal bodies and the posterior part of the thalamus; they give off branches to the plexus choroideus of the third-ventricle and the posterior horn of the lateral ventricle. *Nothnagel* points out, *that with this arrangement, synchronous hemorrhages in the cerebral peduncles and optic thalamus are easily explained.*

In contrast to the vessels of the basal area, which apparently form no anastomoses, the diffusion of the cortical vessels shows numerous anastomoses. Not only do the arteries of one hemisphere communicate in this way, but also those of both halves of the brain (connections of the aa. cerebri posteriores) (*Duret*).

Heubner showed experimentally, that the resistance that opposes the filling of the anastomoses in the soft cerebral membrane is far less than that found in the filling of the finer pia network and the brain itself.

Here, then, a prompt substitution of one artery by another seems possible.

Vascular Supply of Individual Parts of the Brain (*according to Merkel*)

Corpus callosum: branches of the a. cerebri anterior.

Nucleus caudatus: head—branches of the a. cerebri ant.
middle—branches of the second centimeter of the a. cerebri media.
tail—branches from the ramus communicans posterior.

Nucleus lentiformis: a. cerebri med.

Capsula interna: anterior part from the beginning of the a. cerebri media or anterior posterior part from the a. choroidea or the ramus communicans post.

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[1] *The fact that precisely the cerebral arteries are so very predisposed to rupture in arteriosclerotic diseases, is due to their anatomic structure.* We have received important information on this topic from the fundamental investigations of *Bonnet and Triepel.*

"All arteries which may be weighed down or compressed by alternately filled, or movable organs, as for instance intestinal arteries, art. spermat. int., etc., or those which have to carry away the blood in spite of certain resistance due to compression from contracting muscles, like the arteries of the extremities, are furnished very richly with muscles. If they are displaced by movements in their vicinity, their adventitia is especially rich in elastic elements especially longitudinal, elastic nets of fibers. *On the other hand, the arteries in the rigid skull cap show an entirely different and peculiar structure: wide calibre, strikingly thin wall with a very thick elastica interna distinguished by longitudinal supports, whereas for the rest, they have very few elastic elements.*"

Capsula externa: by branches of the second centimeter of the a. cerebri media.

The main artery of the caudate nucleus passes upwards at the boundary between the external capsule and the lenticular nucleus.

Thalamus opticus: The region of the tuberc. anterius is supplied by the larger branches of the ramus communicans posterior, likewise the commissura media. The posterior half receives branches from the a. cerebri posterior.

Tractus opticus: Little branches from the trunk of the carotis, the ramus communicans posterior and the a. choroidea.

Chiasma and nervus opticus: Branches from the trunk of the carotis, from the a. cerebri ant. and the ramus communicans ant. and post.

Corpora quadrigemina: Branches from the a. cerebri post. and cerebelli superior.

Pons: Medial branches from the a. basilaris, from the lower boundary to the locus cœruleus. Lateral branches from the a. cerebelli superior.

Medulla oblongata: Medial branches from the aa. spinales and vertebrales from the beginning of the twelfth cerebral nerve to the horizontal plane of the lower boundary of the pons. Branches of the neighboring arteries, which rise in the nerve roots to the nuclei in the floor of the ventricle. The pyramids and olives receive special lateral branches from the aa. spinales ant. and vertebrales. The lateral column and the corpus restiforme receive branches from the a. cerebelli inf. post. The funiculus gracilis and the margins of the calamus scriptorius are supplied by branches of the a. spinalis post. The lateral parts of the floor of the fourth ventricle receive branches from the a. plexus choroidei.

The *venous* blood collected in the cerebral *veins*, flows off in the large venous sinuses.

Whereas in the fundamental investigations of *Heubner* and *Duret*, the suitability of the *arrangement of the cerebral vessels* to oppose the fluctuations in blood pressure is repeatedly emphasized, we may say, to-day, on the basis of the important investigations of *Otfried Müller*, that the brain possesses in its *vascular nerves* the most important means for the regulation of its supply of blood. *Otfried Müller* rightly emphasizes the fact, that the concept, that our most important and most finely differentiated organ is, with respect to its circulation, helplessly delivered over to the fluctuations of the general blood pressure, is difficult to grasp, in view of the careful regulation, which the kidneys, for instance, possess over their circulation.

Müller in his experiments clings to the experimental arrangement which *Mosso* had used to study the influence of psychic alterations on the flow of blood to the brain. *Mosso* had placed his subjects on a long board, which was supported at its gravitation center, so that both ends were poised freely in equilibrium. If now, in the subject, psychic alterations were evoked, or he fell asleep, the head of the board sank or rose. From these changes in position, *Mosso* concluded that there was an increase or diminution of the flow of blood to the brain.

Otfried Müller used a method of partial weighing which he had worked out personally: if one supports the head of a recumbent subject on a balance, one can, from the changes in weight, which appear under the influence of certain stimulations, draw conclusions as to the corresponding fluctuations in the calibre of the cerebral vessels. *Müller* experimented on men and animals (dogs).

He reached the following important conclusions:

1. Bilateral total division of the vago-sympathetic in the dog, or of the sympathetic in the rabbit, causes considerable increase in the volume of the brain, resp. a considerable increase in the venous blood flowing from the brain.

2. Irritation of the central stump of one of the divided nerves causes a considerable diminution in the volume of the brain, resp. a considerable decrease in the blood flowing from the brain.

3. Both of these changes of circulation in the brain must be interpreted as active processes dependent upon the nervous system, since, in the first place, they appear in point of time before corresponding changes in the general arterial or venous presure, moreover, they are demonstrable only in the brain, and not in other vascular areas (paw, splanchnicus), and finally in some cases they appear without any distinct changes in the general blood pressure.

4. In the vago-sympathetic in the dog, resp. in the sympathetic in the rabbit, therefore, run constrictor fibres for the cerebral vessels, which have a considerable tonus. This tonus is destroyed by bilateral total division of the nerves mentioned and restored temporarily by irritation of one of their central stumps.

5. For the presence of vasomotor nerves in the brain, there is further proof in the influence of chloroform and strychnine upon the circulation. Both drugs attack primarily the vaso-motor center and thence influence fluctuations in the calibre of the cerebral vessels, which are directly opposed to those of the arterial blood pressure.

In like manner, the changes in circulation of the brain caused by the reflex influence of sensory and thermal irritations speak for the presence of cerebral vaso-motors, since, in point of time, they appear before the corresponding fluctuations in the arterial blood pressure. All these reflex fluctuations in the volume of the brain, i. e., of the mass of circulating blood, can not be explained by changes in the venous pressure.

6. In very marked increases in blood pressure the nervous impulses flowing to the brain are overcome, upon occasion, by passive widening of its vessels, so that instead of contraction, dilatation appears.

7. Similar relations are found in the influence of drugs which do not act through the center but attack the vascular wall itself peripherally; for instance, in adrenalin. In small doses, possibly introduced directly into the circulation of the brain, it develops its vaso-constricting influence even over the cerebral arteries. Introduced in large doses into the general circulation, it increases the blood pressure so enormously, that the cerebral arteries are passively dilated.

The cerebral vessels possess, therefore, a nervous self-guidance, which regulates the distribution of blood in the most important organ according to local necessities. The brain possesses in high degree self-government, and can regulate its own circulation depending upon its need.

This reflex regulation of the cerebral vessels is, however, signally different from that subserving the blood supply of other organs. While in the peripheral parts of the body (arm, leg) the contracting vascular reflexes outweigh the dilating ones, the opposite holds good for the brain. *Here the dilating reflexes are more powerful than the contracting.* This is an extraordinarily suitable arrangement; under all circumstances nature provides a plentiful supply of fresh blood to the brain, so that the vital centers should never suffer. If in chloroform narcosis the blood pressure is rapidly lowered, if in hemorrhage all parts of the body give up their contents of blood more quickly, the brain attempts to the last with extraordinary pertinacity to preserve its supply of blood.

Therefore Otfried Müller reaches the important conclusion:

"The conception of a purely mechanical influence upon the circulation of the brain by the fluctuations in the arterial and venous pressure of the general circulation, is untenable. Like the peripheral parts, like the intestines, kidneys, spleen, salivary glands and many other organs, the brain too is subserved by special vasomotor nerves, and only very pronounced increase or diminution in blood pressure can overcome the primary nervous influences."

II. DISTURBANCES IN THE BLOOD SUPPLY OF THE BRAIN

(a) Anæmia and Hyperæmia of the Brain. Cerebral Edema

The diagnosis, *cerebral anæmia*, was made very frequently by old-time physicians. In this there was certainly an exaggeration. But cerebral anæmia presents characteristic symptoms, so that it is justified as a clinical conception (syncope). Its *physiological* foundation is insufficient circulation and nutrition of the brain. Besides the investigations of *Otfr. Müller*, already mentioned, the discoveries of *R. Geigel* have especially increased our understanding of the disturbances in the circulation of the brain.

For the normal functioning of the nervous elements, a plentiful supply of oxygen and the removal of carbonic acid and the products of fatigue are of the greatest importance. An insufficient supply of blood and oxygen bring about at first an *increase* in the excitability of the nerves (*Kussmaul-Tenner's* experiment: after ligation of the carotids, cerebral spasms, loss of consciousness). If the insufficiency of oxygen increases, the nerve excitability is lowered. We know, moreover, that certain centers in the medulla oblongata are excited by blood that is poor in oxygen (respiratory center). In cerebral anæmia, then, *irritation and exhaustion symptoms* may be combined. Besides, there appear various disturbances in the domain of the sensory nerves.

We observe the following symptoms: darkness before the eyes even to amaurosis, contraction of the pupils which changes to dilatation (irritation of the dilator pupillæ through blood that is poor in oxygen), tinnitus aurium, vomiting, headaches and muscular twitchings (irritation of the nerves of the dura and cortical irritation), sleeplessness. In severe cases (especially in acute cases): loss of consciousness, somnolence, syncope. The skin becomes pale and cold. The heart's action is considerably accelerated as a result of injury to the vagus center.

Causes.—Profuse bleeding, chronic poverty of the blood (chlorosis, pernicious anæmia, leucæmia), cachexia, inanition (hydrocephaloid, *Hall*) in children with persistent diarrhœas. Tumors of the brain, ventricular hydrops may cause cerebral anæmia by compression of the organ enclosed in the rigid skull cap. Beside these, acute weakness of the left ventricle (defect in the aorta). *Vasomotor spasm of the cerebral vessels in nervous people.*

In cases of too rapid emptying of ascitic fluid, of puncture of an ovarian cyst, of rapid parturition or the removal of large abdominal tumors, the blood may collect in the temporarily paralyzed vessels of the abdominal cavity (hemorrhage into the abdominal vessels in the sense of *C. Ludwig*). Quite similar conditions exist in the collapse due to paralysis of the vasomotor center in the medulla oblongata (*Romberg and Pässler*).[1]

But an acute cerebral anæmia may be caused also by sudden fright and psychic excitement (see foot-note).

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[1] Violent diarrhœas and purging, however, act in a similar manner. The flow into the abdominal vessels makes the circulating supply in the brain insufficient.

On the other hand, the syncope may be evoked by influences which work directly on or through the nervous system, e. g., sudden severe abdominal pain or pain in the region of the heart. The hypothesis of a direct vagus influence does not explain the matter, since no syncope follows the same irritation if the person is under the influence of an anæsthetic. *Gowers* therefore assumes in a very interesting article (The Borderland of Epilepsy, translated by Schweiger, Vienna, Deuticke, 1908) that the loss of consciousness is caused by an influence on the sensory cortical areas and by indirect influence on the medulla oblongata.

Thus the fainting of many persons at the sight of blood must be of emotional origin. It is not evoked by the most realistic representations. (According to Gowers there are connections between loss of consciousness and petit mal.).

Cases of loss of consciousness due to diminution in calibre of the cerebral arteries, as a result of arteriosclerosis and syphilitic endarteritis, are discussed in another place.

It is possible besides that cases of acute cerebral anæmia may be brought on by mental overexertion (cf. biography of *H. v. Helmholtz*).

Prognosis.—It is usually favorable even when loss of consciousness persists for a long time. But in a few cases death has been reported. In chronic cerebral anæmia the fundamental disease determines the prognosis (arteriosclerosis, cardiac weakness).

Treatment.—In the ordinary fainting spell, we see that the phenomena appear more mildly, resp. disappear rapidly, if we place the patient in a horizontal position, with his head low. Thus, more blood is carried to the brain. *In cardiac weakness:* camphor, wine, coffee. Cutaneous stimulations are also useful (sprinkling the face with cold water, letting the patient smell vinegar, or salammoniac).

In severe general anæmias with chronic cerebral anæmia, iron, arsenic, fresh air and overfeeding should be included in the treatment. Treatment of digestive disturbances is important. The English physicians recommend the administration of *phosphorus* in fainting of neurasthenics.

In *cerebral hyperæmia* one can distinguish so-called *congestive hyperæmia and passive hyperæmia.* The latter results from general congestion in affections of the lungs and heart, in sinus thrombosis, in tumors (pressure on the *vena magni Galeni*, usually combined with hydrocephalus internus). As we have already said in cerebral anæmia *in case of venous congestion the arterial flow to the brain is obstructed on account of the brain's rigid enclosure.* The consequences of passive hyperæmia are, therefore, quite like those of cerebral anæmia: headaches, apathy, vertigo, delirium. The patients, however, do not look *pale*, but cyanotic. The veins in the neck are quite prominent.

The **diagnosis** of a *congestive cerebral hyperæmia* is more difficult. If one does not wish to assume the presence of a cerebral hyperæmia, in persons with so-called plethora or polycythæmia hypertonica, or with veins "swollen during anger," the diagnosis stands on a weak foundation at best. On the basis of *Otfried Müller's* investigations, we are by no means justified in concluding that a cerebral hyperæmia is present from an intensely red "swollen head," nor is the assumption of congestive cerebral hyperæmia after a large meal and indulgence in alcohol, in hypertrophy of the heart, in cessation of the menses, free from objection.

So experienced a clinician as *v. Leube* says: "In any case an active hyperæmia as such can not be diagnosed in my opinion. To be honest, I must confess, that after thirty years practice, not one case is known to me in which I could diagnose an active cerebral hyperæmia with certainty. Whoever concludes on cerebral hyperæmia from redness and heat in the face presupposes a condition of the circulation within the skull that he can not prove."

The possibility of a congestive cerebral hyperæmia can not, of course, be put aside. Perhaps *ophthalmoscopic examination* may be of assistance here. As any rate it is advisable, in suspicious cases, to look for a *contracted kidney* (polycythæmia hypertonica) (hypertrophy of the heart, examination of the urine!).

Treatment.—For the so-called congestive rush of blood to the head in nervous people, a raised position of the head is to be recommended. Cool applications to the head. Derivation by hot baths to the hands and feet (possibly with mustard flour). In *full-blooded* persons: bleeding.

Forbid: coffee, alcohol, full meals. In fat and full-blooded persons treatment at baths and diet (Marienbad, Karlsbad, Kissingen, Bernburg), hydrotherapy. In patients with high blood pressure (above 150 mm. Riva Rocci) the careful employment of *nitroglycerin* should be attempted (in 1% alcoholic solution; several times daily *one* drop, gradual increase and persistent use).

Cerebral Edema

Edema of the brain may be suspected, but can not be clinically diagnosed with certainty. Poor circulation in the brain and toxic influences of various kinds may cause the same phenomena. Cerebral edema may be the result of *inflammations or congestions.* General, inflammatory edema is observed in infectious diseases, toxic edema in nephritis (uræmia. *Traube*). Passive edema has the same ætiology as passive hyperæmia, The older physicians frequently spoke of an *apoplexia serosa*, some younger ones, of *meningitis serosa.* It is certain that frequently there is confusion with cerebral edema. But it must be emphasized that *Quincke* could trace, without a doubt, several quite curious cases to an *idiopathic hydrocephalus*, or to a so-called *angioneurotic* cerebral edema. Some of the cases of so-called pseudo-tumor of the brain may belong here also.

(b) Cerebral Hemorrhage (Apoplexia Sanguinea, Hæmorrhagia Cerebri) and Cerebral Softening (Encephalomalacia) after Occlusion of the Vessels (Endarteritis, Embolism, Thrombosis)

Definition and Ætiology.—In these diseases it is a matter of a *focal affection* of the brain. In *cerebral hemorrhage*, a vessel ruptures and there is a flow of blood into the brain (hæmorrhagic focus); in cerebral softening as a result of vessel closure, a softening focus is formed (encephalomalacic focus).

The phenomena of a mostly transitory diffuse cerebral injury, which leads above all to *disturbance of consciousness*, are common to both affections. A state of deep coma may result from this. The suddenness of the appearance of these general symptoms, justifies the designation, cerebral stroke, apoplectic attack, apoplexy. The designation apoplexy (ἀποπληsία from ἀποπλήττειν—knocking to the ground) was used by the ancient physicians for every suddenly appearing loss of consciousness or sudden paralysis.[1]

We shall now discuss *cerebral hemorrhage.*

A spontaneous, non-traumatic, cerebral hemorrhage is the result of a vascular rupture in the smaller or smallest cerebral vessels. It mostly affects persons who have passed middle age and whose cerebral vessels have suffered atheromatous or sclerotic changes. But fatty and hyaline degeneration, as well as syphilitic disease may lead to cerebral hemorrhage. Cerebral hemorrhage appears as the result of a disease of the vessel wall, which can no longer hold out against the increased or normal pressure of the blood.

As *Charcot* (1868) was the first to show, so-called *miliary aneurisms* frequently form in the cerebral vessels as a result of arteriosclerosis (diameter of 1 mm. and more). They later become the source of the hemorrhage. These small aneurisms may be seated in especially great numbers in the vascular ramifications ascending to the region of the corpus striatum and optic thalamus. But there are also cases of cerebral hemorrhage, in which no such aneurisms exist. It must further be emphasized that the condition of the palpable peripheral vessels affords the examiner no certain indication as to the condition of the cerebral vessels. In pronounced peripheral arteriosclerosis, there need be no great change in the cerebral vessels, and the opposite of this is, of course, also true.

Arteriosclerosis in particular furnishes the predisposition for cerebral hemorrhage. The same ætiological factors play a rôle: *wearing out*, especially at advanced age; *infectious and toxic* influences (alcoholism, gout, syphilis, chronic nephritis). In chronic nephritis, the increase in blood pressure serves to wear out the vascular walls; at the same time, the high blood pressure causes the degenerated vascular wall to rupture more easily. *Aortic insufficiency* also leads, by its constant fluctuations in blood pressure, to arteriosclerosis even in youthful persons.

The cause of apoplexy in *youth* is not rarely the result of a *nephritis* or an *aortic insufficiency.* But in apoplexy of young persons, one must think also of *syphilitic* changes or an *embolism.*

To give an idea of the importance of degeneration of the vascular wall for cerebral hemorrhage, it is necessary to recall that, according to *Volkmann*, not less than a 14 fold increase in the internal pressure is necessary to cause a rupture of the normal carotis.

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[1] *Boerhaave* says: "Apoplexia dicitur adesse quando repente actio quinque sensuum externorum, tum internorum, omnesque motus voluntarii abolentur, superstite pulsu plerumque forti et respiratione difficile, magna, stertente, una cum imagine profundi, perpetuique somni."

"Apoplexy is said to exist, when the function of the five external senses as well as of the internal ones, and of all voluntary motions is suddenly lost, when the pulse is intermittent and mostly strong, the respiration heaving and stertorous, resembling the picture of a deep, permanent sleep."

If the vascular wall is diseased, every transitory increase in the heart's action and the blood pressure may become dangerous: physical over-exertion, psychic excitement, a full meal, indulgence in spirituous liquors, a cold bath, severe straining and pressing during a bowel movement, coughing, laughing, vomiting, coitus, etc.

Why are just the athero-sclerotically diseased cerebral arteries so predisposed to rupture? The answer to this important question is furnished by the fundamental investigations of *Bonnet and Triepel* into the structure of the arterial walls. In contrast to the arteries of the extremities with their strong muscularis and adventitia, the cerebral arteries show strikingly *thin* walls with relative poverty in elastic elements. We must see in the different conditions of the arteries in various vascular regions, a result of adaptation to their environment (overburdening, tearing, pressure on the arteries of the extremities in movements; location of the cerebral vessels in the rigid protecting skull cap) (cf. introduction of this division).

Whoever has experienced one attack of apoplexy is, of course, always in danger of another.

Unquestionably, there are families who, owing to a congenital predisposition (especially poor wearing quality of their vessels), suffer for several generations from apoplexy. It is possible that *hereditary syphilitic* noxæ play some part here also. The older physicians also assumed a *habitus apoplecticus* (undersized build with short thick neck and reddened bloated face). This habitus resembles the appearance in *polycythæmia hypertonica* (so-called Geisböck's disease, which probably is closely related to nephritis). In such individuals, predisposition for cerebral hemorrhage as a result of changes in the kidneys must therefore be thought of.

In recent times, so-called *late traumatic apoplexy* has been much discussed, that is, the supervention of a cerebral hemorrhage a *considerable time* after a cranial traumatism.

According to *Duret and Bollinger*, late traumatic apoplexy is supposedly due to the fact, that as a result of temporary compression of the cranium, the cerebro-spinal fluid is displaced and in certain parts (vicinity of the aquæductus *Sylvii* and the fourth ventricle, the cortex) gives rise to a cerebral softening. The process may lead not only to direct softening, but also to degeneration of the vessels, which may in turn be the cause of an apoplexy.

Langerhans, in contradistinction to *Bollinger*, simply assumes a traumatic aneurismal formation.

The following is also assigned as a cause of apoplexia cerebri: cessation of hæmorrhoidal hemorrhages (*Oppenheim*). In the cases observed, however, *arteriosclerosis* was present. By this simultaneously existing arterio-sclerosis, we must probably also explain the so-called vicarious cerebral hemorrhages (in stoppage of the menses).

The hæmorrhagic diathesis more frequently leads to meningeal extravasation, than to deeper cerebral hemorrhages.

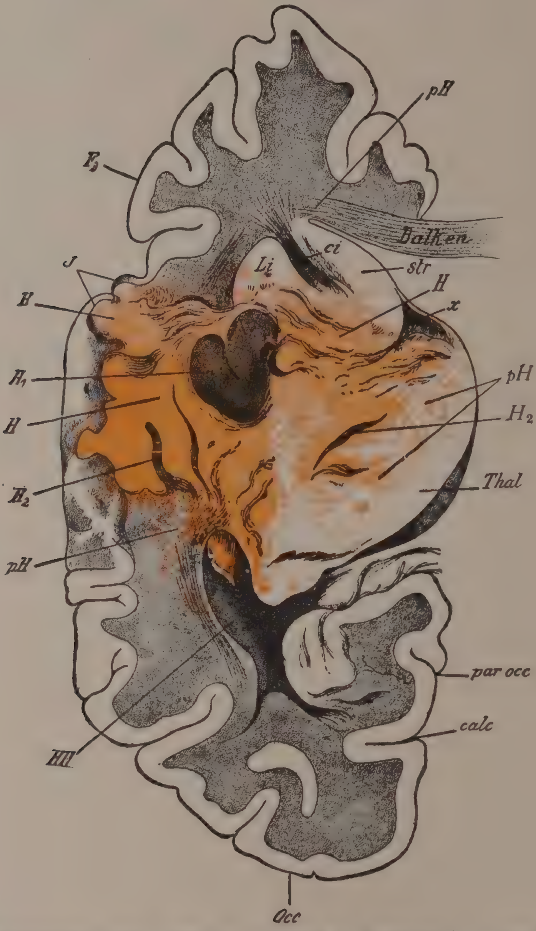

FIG. 179.—Hæmorrhagic focus 5 days old. (*From v. Monakow.*) Caused by rupture of a miliary aneurism in the lenticulo-optic artery. Perforation into the lateral ventricle (at x). Horizontal cut through the left cerebral hemisphere. *H* 1, primary firmly coagulated blood clot. *H*, a later blood extravasation with several firmly coagulated stripes (H2); *pH* punctate and lineaform secondary extravasations in the farther periphery of the primary focus, caused by diapedesis; *Thal*, optic thalamus, edematous in a high degree and pushed over the median line to the left; *ci*, anterior limb of the internal capsule, almost normal; *Li*, putamen, external layer of the lenticular nucleus; *HH*, posterior horn, contains several cruor masses; *J*, insula; *F*3, third frontal convolution; *Occ*, occipital pole; *par occ*, fissura parietooccipitalis; *calc*, fissura calcarina.

The *favorite seats* of spontaneous cerebral hemorrhages, are the large central ganglia (*corpus striatum and thalamus*) and the internal and external capsule, therefore the *vascular realm of the arteria fossæ Sylvii.* Here also miliary aneurisms are most frequently found. Hemorrhages in the medulla oblongata are least frequent.

Pathologic Anatomy.—In most instances it is a matter of one hæmorrhagic focus. Multiple hæmorrhagic foci, appearing simultaneously, are rare.

The size varies considerably; usually the focus is no larger than a hazel nut or a walnut. But very small and very large hemorrhages have also been observed. Thus, the larger part of a hemisphere may be destroyed. In the immediate vicinity of the hemorrhage the substance of the brain is destroyed; a bloody pulp is formed. At the periphery of the focus, the tissue is tattered and mostly infiltrated with blood. One sees small torn vessels. After some days the vicinity of the focus appears edematously drenched. The edema is colored yellow by the coloring matter in the blood (lemon-colored edema).

The *cerebral ganglia* and their surroundings, the *centrum ovale* above the ventricles are most frequently the seat of the hemorrhage. The other more important parts of the brain are affected in the following order of frequency: *Convolutions of the cerebrum, cerebellum, pons, pedunculi, medulla oblongata, cerebral membranes.* Hemorrhages in the neighborhood of the ventricles may break into the ventricles. On the other hand, rupture toward the surface of the brain (convexity or base) is rare. The breaking through of the hemorrhage into the ventricles, hemorrhages in the pons and the medulla oblongata are usually fatal.

But in many cases, the attack passes off, and after a shorter or longer time, there is a repetition. In the larger hemorrhages, the consequences of spacial restriction become manifest: the dura mater is tightly stretched, the sulci are wiped out, the surface is flattened.

F. v. Niemeyer was probably the first to call attention to the resistance, which falx and tentorium offer to the spread of cerebral pressure. Thus in a hemorrhage of one hemisphere, the surface of the brain may be more flattened on the side of the focus than on the other. In hemorrhages beneath the tentorium, the surface of the hemisphere of the cerebrum may not be flattened (*Liebermeister*).

If life is spared the *hæmorrhagic focus* shows certain changes. At first the blood coagulates in the hemorrhagic focus. The serum is pressed out, and removed by the blood and the lymph. In this way, even large foci may be considerably contracted; the cerebral pressure, originally exerted by them, disappears. Leucocytes absorb the red blood corpuscles and the free coloring matter of the blood. The cerebral detritus disappears by *autolysis*, or is partly also removed by the leucocytes. The focus becomes a fluid pulp. The color changes too; it becomes brownish-red, later rust color shading off to yellow (Fig. 179). In the neighborhood, there is glia production; in this way a small focus may disappear altogether. In its place forms a pigmented connective-tissue cicatrix (*apoplectic scar*).

Larger foci are encapsuled by regional proliferation of connective tissue and glia; the space that has been cleared by absorption is filled with serum (*apoplectic cyst*). The size of the cysts varies between that of a pea and of a walnut. In one case *Kaufmann* observed a cyst as large as a hen's egg in the substance of a hemisphere. In the vicinity of a cyst, the substance of the brain is often *atrophic.*

In lesion of the motor tracts secondary descending degeneration (*Türck*) develops.

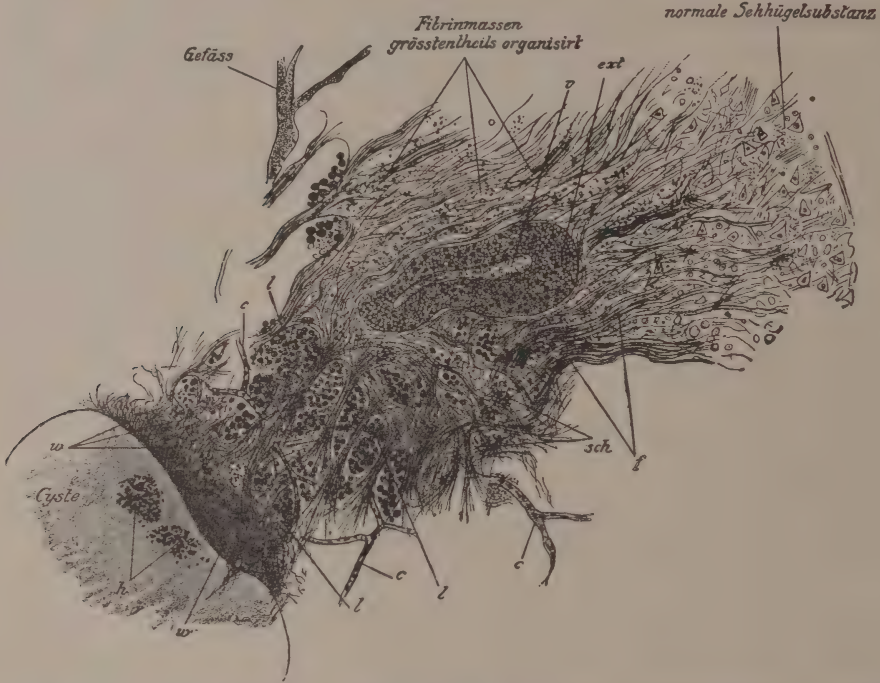

FIG. 180.—Segment of a 2 years' old hæmorrhagic focus (cyst) in the optic thalamus. (*From v. Monakow.*) Cyst, cystic wall and transition into normal optic thalamus tissue, 200 times magnified. *w*, cyst wall, fibrous; *v*, blood vessel with extravasated blood in the adventitia (ext); *sch*, pigment spots, hæmatoidin and hæmosiderin crystals; *f*, connective tissue (organized fibrin masses); *c*, capillaries; *l*, granular cells, rolled together red blood corpuscles and blood platelets, enclosed in lacunar spaces; the walls of the latter consist of organized fibrin; *h*, transformed coagulated blood sticking to the cyst wall. In the cut: *Gefaess*, vessel. *Fibrinmassen groesstentheils organisirt*, fibrin masses organized to a great extent; *normale Sehhuegelsubstanz*, normal substance of the optic thalamus.

Symptomatology.—We distinguish *diffuse* and *focal* symptoms. The diffuse phenomena correspond to the various degrees of disturbance in consciousness. The more quickly a hemorrhage appears, the larger the hemorrhage, the more outspoken is the effect on consciousness (apoplectic seizure, stroke). These diffuse phenomena may soon pass away in contrast to the focal phenomena, which, being the result of the local cerebral destructon, are lasting. The most frequent result of an apoplectic seizure is *hemiplegia.*

The symptom complex begins mostly with the so-called *apoplectic attack.* The patient loses consciousness more or less completely, he falls to the ground "being felled by the stroke." But there are cases in which the real apoplectic attack does not develop and the patient himself diagnoses his sudden paralysis. A *severe* attack leads to coma, loss of all voluntary movements and of conscious sensation. *In this condition, all reflex movements may be lost.* The patient's excretions are discharged involuntarily, occasionally, too, there is retentio urinæ. The *urine* may for a time contain sugar and albumen (distant effect upon the medulla oblongata). The *face* looks pale red, "bloated." *Breathing*, as result of paralysis of the soft palate, is mostly heavy. The *thorax* on the paralyzed side may in breathing remain obviously behind. *Cheyne-Stokes* respiration is frequently observed. In expiration, the cheeks are puffed out (relaxation of the buccinator). The *pulse* is usually full and slow. The carotids pulsate visibly.

The attentive observer may often, even during the coma, recognize a unilateral paralysis; the face is drawn to one side, the extremities of one side are totally flaccid and upon strong sensory stimulation there are no protective movements on the paralyzed side.

Convulsions and direct muscular contractions are rare; in *severe hemorrhage* they are the expression of cortical irritation.

The *bodily temperature* may be abnormally low. A *considerable increase in temperature is usually an unfavorable sign.*

Prévost pointed out that head and eyes may exhibit forced movements towards one side, "as if the patient would look towards the seat of the cerebral lesion" (déviation conjugée). The center for the conjugated eye movement probably lies in the lower parietal lobe. The condition of the *pupils* varies considerably. In some cases they are dilated, in others contracted, often unequal. There may temporarily be reflex pupillary rigidity to light.

Prodromes may precede the attack: headaches, dizziness, slight pareses, disturbances in sensation. Several times I observed that an apoplectic attack was preceded by a *feeling of numbness* in one hand. *Retinal hemorrhages* may have a premonitory significance.

The attack may occur almost instantaneously (apoplexie foudroyante), but it may also develop gradually. The duration of the attack usually varies within several hours. It rarely lasts for days. *The longer the apoplectic seizure lasts, the worse the prognosis usually is.*

The *cause of the attack* must be sought in the increase in intra-cranial pressure, and the *cerebral anæmia* resulting from it. Of course, we must admit that there is a great difference between a simple fainting spell and the coma of apoplexy. The disturbance in consciousness corresponds rather to the loss of consciousness in an epileptic attack. In severe attacks, therefore, one must think of a sort of commotio cerebri (etonnement cérébral).

The *behavior of the tendon reflexes* is peculiar and not easily explained.

According to the general theory, one would expect in so severe an injury of the general function of the brain (interruption of the influence of the will and loss of the reflex inhibiting factors) the reflexes to be increased, on one side, at least. But this is not the case. *The coma is often accompanied by total loss of the reflexes.* The reflex conductivity must, therefore, be for the time being totally destroyed. As the coma of the severe attack disappears, the reflex activity is gradually restored, and by careful examination the presence of *considerable increase in the reflexes on the paralyzed side* may be demonstrated early (decrease or loss of the physiological reflex inhibition through the cerebral lesion).

The *cutaneous reflexes* appear to be diminished or entirely lost, especially on the paralyzed side. *On the basis of this fact the seat of the paralysis may be established even in stuporous patients by testing the cremaster and the abdominal reflexes.* The different behavior of the tendon and cutaneous reflexes may possibly be explained by the fact, that the arcs of the cutaneous reflexes are located at a higher level than the arcs of the tendon reflexes. They go through the brain. Their absence, therefore, is to be traced back to an injury of the reflex path. Of especial practical importance for the disturbance in reflex inhibition on the paralyzed side, is proof of the presence of the *Babinski reflex.*

If the apoplectic attack does not cause death in the coma, then the severe general phenomena, so alarming to those that surround the patient, disappear gradually and the disease picture is now dominated by the *focal* symptoms. Systematic clinical medicine has for a long time distinguished between *direct* and *indirect* or *permanent* and *transitory* focal symptoms. Anatomically expressed, the question now stands "*what is destroyed in the brain, what is only compressed*, i. e., *temporarily disturbed in its function?*" The answer to this question can be given only by the further course of the disease. Taken all in all, however, and basing our statements on an experience of many cases, we are able to say, with a certain degree of probability in the individual case, that *such* symptoms will disappear in *this or that order*, and that *others* will probably remain permanently.

In the majority of cases, a *hemiplegia* remains as a focal symptom.

But even the hemiplegia occurring after severe attacks may disappear completely in case it was not the result of destruction of the motor tracts, but was simply produced by indirect influence on these tracts (hemorrhage in the vicinity).

In the anatomic introduction we have already pointed out why hemiplegia presents the *typical* form of cerebral paralysis. The cerebral hemorrhage occurs preferably in almost three-fourths of the cases, in the neighborhood of the ganglia of the cerebrum. Corresponding to the course of the motor tracts (crossing of the pyramidal tracts) it is *contralateral.* It strikes, more or less, most of the muscles of the side of the body which is opposite the focus. It shows itself with peculiar distinctness shortly after the awakening from the coma in the *flaccid dropping* of the passively raised *arm, or leg.* If the hemorrhage has broken through into a lateral ventricle, tonic rigidity of the extremities may also be observed (*Strümpell*). We are able to prove the presence of *increase in the patellar reflex*, *Babinski* reflex, (dorsal flexion of the toes, especially of the great toe, instead of the normal plantar flexion consequent upon irritation of the sole of the foot or of the ball of the great toe).

Cremaster and abdominal reflexes on the paralyzed side are weak, or lost. The tendon reflexes on the sound side are also usually increased, though only to a slight degree.

In many patients, we see as soon as we step near the bed which side is paralyzed from the peculiar facial expression. The mouth is distorted and drawn towards the sound side. The corner of the mouth on the paralyzed side droops. The nasolabial fold is flattened out. There is paralysis of the *lower facialis.* The upper facialis (forehead, lid closer) is, in contrast to the peripheral paralysis of the facialis, free of paralytic phenomena. Indications of pareses of the mm. frontal., corrugator and orbicularis palpebr. are, it is true, found in cerebral paralysis also.

The tongue is protruded sideways, drawn towards the paralyzed side by the preponderant influence of the m. genioglossus of the healthy side (hypoglossal paresis).

Aphasia appears in ordinary apoplexy, mostly only as an indirect, *transitory* symptom.

The musculature of the trunk also (respiratory muscles) seems paretic. *Leube* emphasizes the fact that this may cause a predisposition to the development of a pneumonia.

As regards *sensibility*, a transitory, slight *hemianæsthesia* is observed in most cases. Pronounced and lasting hemianæsthesia must awaken a suspicion of a direct focal symptom. Furthermore, one observes disturbances in the muscle sense: "the patient does not know how and where his leg lies."

The *vasomotor* and *trophic* disturbances are to be regarded as *indirect* focal phenomena. The paralyzed extremities may show increase in temperature. *Acute decubitus* must also be viewed from the standpoint of such disturbance (*Charcot*). It attacks the glutæal region of the paralyzed side very quickly, a few days after the apoplectic attack.

If we exclude *changes in the joints*, which appear only because of *inactivity* in the paralyzed limbs, we may possibly interpret as so-called trophic disturbances the rare joint changes in apoplexy that are characterized by swelling, redness and pains. Furthermore, *edema* may be observed on the paralyzed side. It might be traced back to the insufficient movement of the blood and to vascular changes in the paralyzed side.

Since the vaso-motor conducting tract may pass in the vicinity of the sensory path, or, according to *Parhon-Goldstein* in the anterior part of the internal capsule, a *direct or an indirect injury of the vaso-motors* of the paralyzed side must also be thought of.

In occasional cases, a remarkable *muscular atrophy* has been observed in the *paralyzed limbs*, which can not be explained simply by the lack of movement alone. Decrease in electrical excitability was clearly visible, but never E. R. D. *Charcot* thought of a degeneration of the anterior horns, following the descending degeneration of the pyramidal tracts. *Steinert* makes the loss of motor stimulations responsible.

With the return of consciousness, the physician is for the first time given the possibility of a more exact topical diagnosis. But we can not yet say, which of the phenomena will remain permanently and which not (*direct and indirect* focal symptoms). But after several weeks have passed after the onset of the attack, then we can say with greater certainty, which of the symptoms are to be regarded as permanent. But sometimes, even after months, focal symptoms retrogress.

As regards the special localization, we refer to the section of this book written by *Liepmann*.

Here we discuss only the most *important* focal symptoms. It must first be emphasized, that the hemiplegia is not completely regular. Nearly always the arm is more severely paralyzed than the leg, and in the arm itself, the paralysis is more pronounced in the muscles that subserve the movements of the hand. We have already said that the paralysis of the facial strikes only the lower branches. The paralysis of the hypoglossal, of the musculature of the neck and trunk, is less pronounced than the paralysis of the extremities.

This condition may be explained by the varying degree of degeneration found in the individual fibres, but the hypothesis of *Broadbent*, supported by the observations of *v. Leube*, furnishes a very illuminating explanation. According to *Broadbent*, only the muscles that perform voluntary movements with their fine shadings in motion, are innervated from *one hemisphere*.

The muscles of the neck and trunk, most of the muscles innervated by the facialis, the muscles that turn the eye laterally, on the other hand, are innervated by both hemispheres (of course less powerfully by one of them). Muscles that have symmetrical activity in both halves of the body are, according to our experience, only temporarily paralyzed by one-sided foci. The hemisphere, that remains intact, may intervene as a compensating factor.

Furthermore, *v. Leube* points to the fact, that this hypothesis of *Broadbent* furnishes a very good foundation for the fact, that in recent hemiplegias not only the contralateral, but even the same side may evince disturbances in movement.

As a matter of fact, these pareses appear more clearly in the leg than in the arm. Therefore in a hemorrhage of the left hemisphere, there may appear beside the paralysis of the right extremities, also a decrease in the strength of the *left* arm, but especially in that of the left leg.

Possibly, the *associated movements*, which apoplectics exhibit on the sound side when they exert themselves to move the paralyzed side, are also to be explained in this manner. The *tibialis phenomenon* cited by *Strümpell* is also to be explained in the sense of an associated phenomenon. The patient can not perform an isolated dorsal flexion of the foot of the paralyzed side but if he attempts to draw up the entire leg to the trunk, then there appears a strong dorsal flexion of the foot with pronounced tension in the tendon of the N. tibialis anticus. Thus the patient can contract only larger groups of muscles "synergically."

The seat of the hemorrhage is, as has been described, most frequently in the corpus striatum, in the *internal capsule* and the neighboring centrum semiovale. Hemorrhages in the *internal capsule* give rise to a pronounced motor *hemiplegia.* If the seat of the hemorrhage in the internal capsule lies more in the lower part of the posterior division of the capsule, then there appear beside the motor paralysis, sensory and vaso-motor disturbances as well.

If beside the motor hemiplegia there is an oculomotor paralysis of the other side (ptosis, outward rotation of the eye, dilatation of the pupil) a *focus in the pedunculus cerebri* must be thought of.

A *focus in the pons* causes besides hemiplegia on the opposite side, paralysis of the abducens, trigeminal or *facial*, on the same side.

In a *focus in the oblongata* one may find besides hemiplegia of the opposite side, paralysis of the hypoglossal on the same side.

In the larger hemorrhages in the *pontile and oblongata region*, however, death usually follows.

If it concerns an extensive cortical hemorrhage with monoplegias combined with hemiplegia, then there appear (often after weeks and months) *epileptiform* twitchings in the paralyzed extremities and in the face. *Aphasia* also is found rather frequently in such cases.

But *monoplegias* may appear also, if the seat of the hemorrhage is in the centrum ovale, where the fibres for the arm and leg are not near each other. On the other hand, in contrast to central infantile paralysis, hemi-athetosis posthemiplegica in the apoplexy of older people is very rare.

In foci in the centrum ovale epileptiform twitchings and aphasia are naturally absent.

Differential Diagnosis.—Meningitis may lead to coma, and in rare cases, also to hemi-pareses. But at the same time, it is accompanied by the characteristic hyperæsthesia of the entire body. It leads to convulsions and contractures, to paralyses of the eye muscles, and is accompanied by fever. An examination of the background of the eye often discloses *neuritis optica.*

Apoplectiform attacks appear, furthermore, in *multiple sclerosis*, *progressive* *paralysis, in cerebral tumors and abscesses.* The anamnesis and a most careful examination, especially of the background of the eye, soon put an end to all doubt (sclerosis: gray atrophy of the optic nerves; tumor; choked disc).

The differential diagnosis between cerebral hemorrhage and toxic hemiplegia, in cases of *uræmia*, may be difficult. Coma is found in both conditions. Albumen may appear also temporarily in the urine in severe apoplectic attacks, without the presence of a nephritis. On the other hand, the contracted kidney leads also to arterio-sclerosis, and with its arterial degeneration and the increase in blood pressure, produces a tendency to a cerebral hemorrhage. In such a case, one must confess, it may be absolutely impossible to make a certain diagnosis during the coma. One must wait, and first of all try to secure an exact anamnesis.

As soon as focal symptoms appear, and convulsions and vomiting are absent, the diagnosis is usually easy. Absence of the abdominal and cremaster reflexes on one side, and conjugate deviation of the eyes, may early indicate a cerebral hemorrhage, when the flaccidity of the extremities of the one side, is not yet very evident. As is well known, however, transitory hemi-pareses may appear in uræmia also.

The differential diagnostic distinction from *hysterical* paralysis may also be very difficult. In hysteric hemiplegia, there is usually no paralysis of the facialis. *A hysteric paralysis of the facial is, taken all in all, the greatest rarity.* Anamesis, general habitus, absence of coma must help here, but above all, one must be on the lookout for hysteric zones, for hysteric hemianæsthesia, with unilateral disturbance in hearing, smell and disturbances in the field of vision. But most difficult is the differential diagnosis between cerebral hemorrhage and pachymeningitis cerebralis hæmorrhagica. We shall return to this in our discussion of *dural hemorrhages.*

Course and Prognosis.—If the patient does not die during the attack, the rest of the course depends first of all upon whether or not pneumonia develops. The focal phenomena may retrogress even after months. Complete recovery is observed only in very small hemorrhages or in slight embolisms (cf. following chapter), if there is prompt formation of the collateral circulation. If the paralysis was caused only by pressure or by local edema, it may gradually retrogress. "Vicarious" conduction paths may also develop.

Charcot emphasized the fact, that a restitution is not to be expected if the internal capsule is destroyed, but if the hemorrhage is situated in the corpus striatum and the lenticular nucleus, in the majority of cases, a considerable improvement takes place.

If the paralysis persists, there is often a development of *contractures* in the paralyzed muscles. Contractures are especially frequent in hemorrhages in the capsula interna (Charcot). The arm is usually in a position of *flexion and pronation*, the leg in that of extension or flexion, but usually the contracture is in extension. The face may be drawn over towards the paralyzed side. These contractures, therefore, are passive, i. e., the muscles are shortened as a result of paralysis. The increase in the tendon reflexes on the paralyzed side also leads to tonic tension of the musculature. Thus, *Hitzig* observed that some contractures are slight during or after sleep, and appear again only after movements. The paralyzed muscles and nerves respond both to the constant and faradic current. No E. R. D. appears. In occasional cases, however, diminution in the electric excitability has been observed (*Steinert*).

Sometimes, with the return of mobility in the originally paralyzed extremities, *choreic or athetotic* movements appear (post-hemiplegic chorea or athetosis). This is especially the case when the focus is situated in the optic thalamus, or in the posterior part of the internal capsule.

Exact time limits relative to improvement and cure, can not, of course, be given. In general, it must be said, that after every apoplectic attack, the first two weeks are always critical, because of the possibility of a second attack, or the development of pneumonia.

Gradual improvement after severe attacks may continue for months. But whatever has not disappeared in from 6–9 months, must be regarded as a permanent defect. The more quickly after an attack the improvement in motility appears, the more favorable is the prognosis. But there are exceptions to this rule also (cf. the following chapter). It is not at all rare to see a lighter apoplectic attack disappear without leaving any traces, and then suddenly to have the patient prostrated again by another and a very severe stroke.

Therefore we must be very cautious with the prognosis even in apparently slight cases.

The cases in which an apoplectic attack is followed by a progressive decrease in the psychic functions even to *dementia*, are especially sad (cf. the chapter "Arterio-sclerosis of the Brain").

Treatment.—The older physicians *bled* their patients in every case of apoplexia cerebri. Among the clinicians, *Liebermeister* was an adherent of treatment by bleeding in cerebral apoplexy. He recommended it in those full-blooded individuals, whose radials and carotids pulsated strongly; and whose faces were very red. He rejected it, when there was weakness of the heart and in anæmic patients.

But *Trousseau*, who at first bled the patients in all cases, later changed this method entirely. And, indeed, according to the investigations of *Otfried Müller* a considerable influence of the bleeding upon the brain pressure can not be expected.

Nevertheless, especially with patients in whom the blood pressure is high (apoplexy in contracted kidney) there is an impression, that bleeding is not entirely useless. So keen an observer as *H. Geigel* would not remove it entirely from the therapy of cerebral hemorrhage.

It goes without saying, that the person who has had the stroke must be undressed and put to bed. The bowels should at once be emptied by rectal injections. Also it should not be forgotten to empty the filled bladder of unconscious patients. Attention must be paid to the lungs (pneumonia). Considering the possibility of an acute decubitus in severe cases, a water bed is recommended.

Experienced physicians put an ice bag to the head if the blood pressure is high and there is visible congestion; they avoid the use of the ice bag when the blood pressure is low and the patient looks anæmic. *Oppolzer advised* observation of the condition of the jugular vessels to decide whether the cerebral phenomena are due to anæmia or hyperæmia. Geigel would omit the ice bag during the acute attack "up to the moment when the most severe cerebral symptoms tell us, that it is now time to get relief at any price, through improvement of the circulation for the brain, that is about to choke." (Cf. the following chapter for the treatment of cardiac weakness in *embolic* processes.)

During the period following the attack careful *nursing and appropriate nutrition* are the main things. One must see to regular evacuation of the bowels (diet, stewed fruit, rectal injections, mild laxative medicines, rhubarb, pulv. liquiritiæ compos., Cascara Sagrada, etc.).

For fulfilment of the *indicatio causalis*, if a syphilitic arterial disease is present, energetic syphilitic treatment must be instituted (mercurial inunctions 1–2 ʒ pro die and the internal administration of iodide of sodium 30–45 gr. pro die).

Small doses of iodide of sodium are useful also in *arterio-sclerosis.* According to the investigations of *Müller* and *Inada* the iodides diminish the internal friction of the blood (viscosity) and thus facilitate better circulation in the brain. In cases with *high blood pressure* in arterio-sclerosis, in contracted kidney, the cautious use of *nitro-glycerine* is recommended.

Concerning the prevention of contractures, it is important to remember the investigations of *H. Munk. Munk* proved in apes, that the hemiplegic contracture is mainly a result of inactivity, and that it can be avoided by systematic passive movements. Therefore one must agree unreservedly with *Geigel*, in recommending in suitable cases, to begin 2–3 weeks after the attack regular stretching of the muscles, that incline to contracture. The stretching should be done twice a day for about 5–10 minutes, and this treatment should be continued, if necessary, for months. *It may be useful to combine the stretching with massage.*

The residual paralyses are treated with peripheral *faradization*, or *galvanization*, and hydro-therapeutic measures. I should not advise direct cerebral galvanization. In any case, one must not begin too soon with the electrical measures mentioned, since any irritation of the brain may prove injurious for some time after the attack. One should wait patiently for a few weeks.

Hydriatic treatment also should not be begun until about the fourth week after the attack. One should begin with systematic ablutions of the paralyzed extremities (beginning with lukewarm water, and gradually cooling it down to the temperature of the room. Possibly with the addition of arnica) once or twice daily. If they are well borne, then two weeks later one should try an indifferent bath (94–96° and lasting five minutes).

The baths are gradually to be lengthened, and the patient made to perform movements in the bath; the patient may be left in the bath even for half an hour. (Contracture inhibiting influence of the bath, aided by simultaneous massage.) Two to three baths should be given per week.

Erben and *Matthes* have recently advised hot compresses to the head to prevent the severe vertigo which sometimes appears at the first attempt to walk.

It is self-evident, that for a patient who has once had a cerebral hemorrhage, the entire remainder of his life must be regulated prophylactically. Most patients observe the physician's orders willingly, for there are no more obedient patients than hemiplegics who observe a gradual improvement in their condition.

So that the patient shall not lose courage, the experienced physician will know how to find some measures, that work by suggestion (certain additions to the baths: as salt, extract of pine needles). CO_2 baths have also been recommended enthusiastically. But we must be very careful with such cool baths, as they increase the blood pressure. At any rate they are suitable only in some cases and must be given very weakly charged. They certainly are not suitable for the treatment of the first stage of hemiplegia.

For later treatment the following places are suitable: Wiesbaden, Nauheim, Schlangenbad, Marienbad, Oeynhausen, and others—namely, places that are suited for arterio-sclerotics. At any rate one should avoid places that are higher than 500 m. for people who have had even the lightest attack.

A correct general treatment will have to consider dietetically and prophylactically everything demanded by the treatment of arterio-sclerosis.

(c) Cerebral Softening through Vascular Occlusion

(*Embolism, Thrombosis, Endarteritis*)

Definition and Ætiology.—Embolic occlusion and autochthonous coagulation of the blood (*thrombosis*), besides the *endarteritic* processes are the causes of the sudden or gradual closure of the cerebral vessels.

As we saw in the introduction to this chapter, while discussing the vascular supply of the brain, the arteries of the basal region show a deficient collateral development (so-called "endarteries" in the meaning of *Cohnheim*). On the other hand, the cortical arteries show manifold anastomoses. The cerebral cortex, therefore, is better supplied collaterally than the large ganglia and the centrum ovale.

This relation is best illustrated by the fact that upon occlusion of one *a. fossæ Sylvii* (five main branches for the cerebral cortex; besides this it supplies the largest part of the nucleus caudatus, the nucleus lentiformis, the capsula interna, and a part of the optic thalamus) disturbance in the nutrition of the cortex may be entirely absent (*Ed. Kaufmann*).

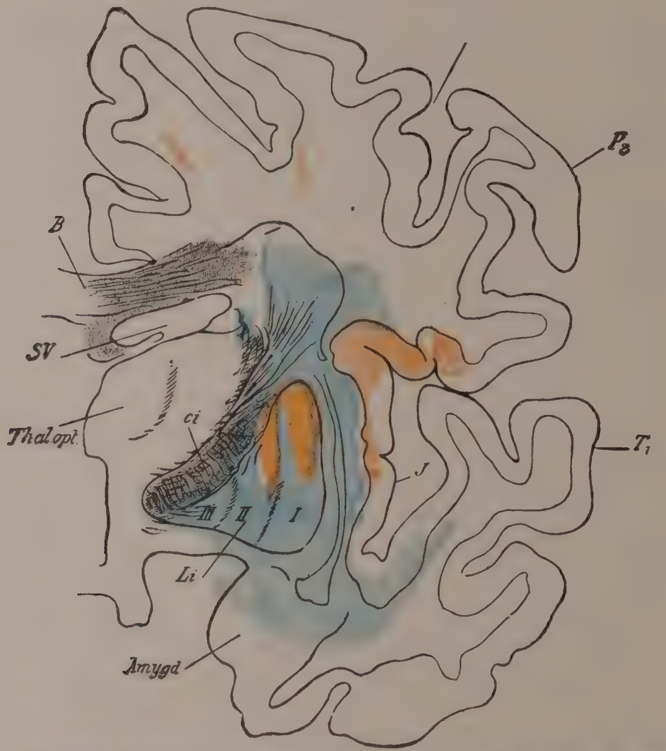

FIG. 181.—Extension of the softening focus in recent embolism of the right art. carot. int. in a 40-year-old person. Frontal cut through the cerebrum in the region of the anterior central convolution. (*From v. Monakow.*) The softened places are rendered in blue, the intensity of the color corresponds to the degree of the necrosis; the very dark spots are liquid. The hemorrhagic, resp. the extravasated places are red; *B*, corpus callosum; *Amyg*, amygdala; *Thal opt*, optic thalamus; *Li*, lenticular nucleus; *J*, insula; *Tl*, first temporal convolution; *P2 Gyr.*, gyrus supramarginalis; *Jp*, interparietal fissure; *ci*, internal capsule; *SV*, lateral ventricle.

The emboli mostly arise from the left side of the heart (endocarditis). If the foramen ovale is open, they may also come from the right side of the heart (crossed embolism). Furthermore, thrombi in the *auricles of the heart*, in arterio-sclerotic vessels, in *aneurisms* may be the cause of emboli.

Thrombi are especially prone to form in the arterio-sclerotic aorta, in the bifurcation of the carotids, also in the pulmonary veins (in cavities and gangrene). The emboli mostly remain clinging to the bifurcations of the vessels.

In athero-sclerosis, as well as in toxic-infectious disturbances of the cerebral vessels, *thromboses* may arise in the vessels themselves.

Thus, *acute infectious diseases* (typhoid, diphtheria) lead to *local* injuries of the intima, which then become the cause of thrombotic formations. *An embolism may emanate from any thrombus.* Most frequently the *a. fossæ Sylvii* (or its branches) is affected by an *embolism*, and moreover, the left more frequently than the right. The embolic occlusion of a *carotid* or a *vertebral artery* is rare. The *thrombotic* occlusion of the cerebral vessels is not so frequent as the embolic. But precisely in arterio-sclerotic dementia, multiple thrombosis of small, even of the smallest cerebral vessels plays an important part.

The consequences of the occlusion of a cerebral artery are dependent on the extent of the collateral connections. Thus it is of importance, whether the vessel is on *one side, or on the other, of the circulus arteriosus Willisii:* the occlusion of a carotid, for instance, need evoke no severe, or at least permanent, disturbances. If, on the other hand, the *trunk of the a. fossæ Sylvii* is closed, its *entire* vascular region may be softened.

If, after occlusion of a cerebral artery, there is no collateral supply to the shut-off region, *ischæmic necrosis* and softening will result. If the ischæmic region is permeated with blood from the vicinity, we speak of *red cerebral softening*. If the coloring matter in the blood is transformed, there arises the so-called brown or *yellow* softening. If no blood has entered the dead region, there is so-called *white softening*. It is evident, that for the origin of a softening focus it is important, whether the occlusion results *suddenly* or *gradually*, whether the *heart* is strong or has been weakened.

For if collaterals are present, they will be able to dilate, when there is good blood pressure and the (*thrombotic*) occlusion is gradual, so that the softening focus will be far smaller than when there is sudden occlusion and poor cardiac action. The conditions are quite analogous to those in coronary sclerosis of the heart (cf. introduction to this chapter). (In any case, the concept of endarteritis needs revision by experiments also with regard to the brain.)

If the embolus has its origin in a gangrenous focus of the lungs, or in a purulent focus, gangrenous softening or a metastatic abscess develops. (*Cerebral abscess in bronchiectasia.*)

The size of the *encephalomalacic* foci may, of course, vary considerably. Just as in hemorrhage, it may come to formation of *scars and cysts*. With numerous old, small foci, the brain may appear perforated like "Sweitzer cheese." If the foci lie near the surface, deep depressions may form.

The anatomic picture may resemble the changes designated by *Ferrard* as *encéphalite chronique sclérosique des vieillards* or *lacunes et desintégration cérébrales* (*Schumann, Kaufmann*). (Fig. 182.)

Symptoms.—Even the *anatomic* picture points to the fact that the symptoms are dependent 1. on the sudden occlusion in itself (attack), 2. on the phenomena of functional loss, the result of the softening, but there are cases of so-called chronic softening of the brain in which, without any real attack, one symptom of functional loss after another appears.

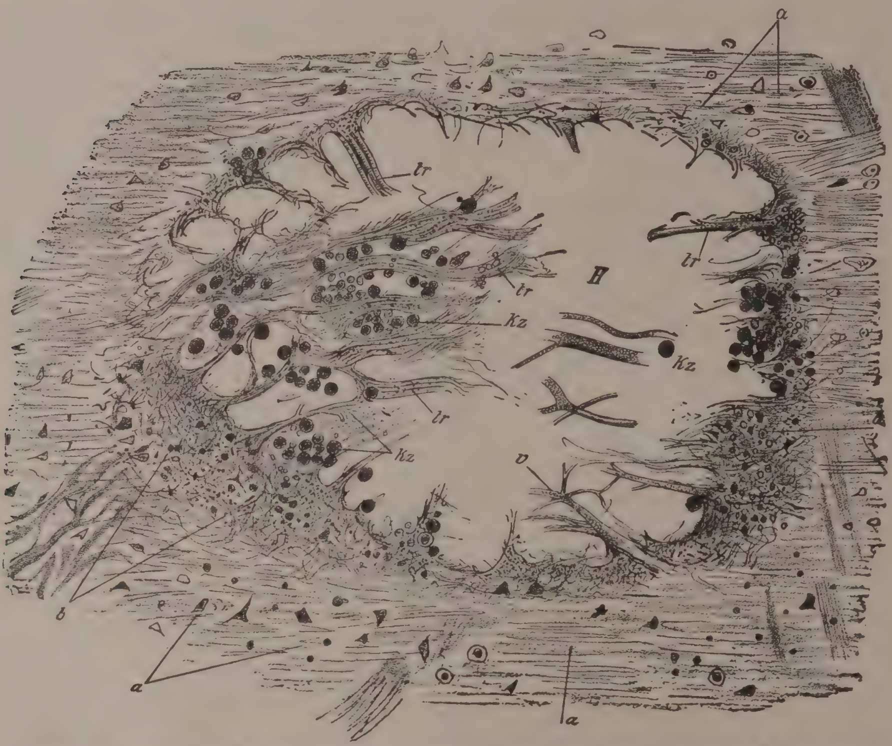

FIG. 182.—Small old ischæmic softening focus in the right optic thalamus of a 70-year-old man. (*From v. Monakow.*) Magnified 300. *a*, normal substance of the thalamus; *b*, shrunken (sclerotic) wall of the focus; it contains proliferated glia cells, spindle cells, etc.; *H*, cavity of the focus filled with serous fluid and detritus; *Kz*, granular cells; *tr*, shrunken trabeculas, going down into the cavity and consisting of capillaries, transformed glia fibres and necrotic nerve elements; *v*, small vessels.

The apoplectic attack in a case of vascular occlusion can not in itself be distinguished from that in hemorrhage, but *with embolism it is decidedly rarer and shorter. Prodromal symptoms* are mostly lacking, or they correspond to the general cerebral phenomena, well known in arterio-sclerosis of the brain (headache, vertigo, psychic depression).

The intensity of the attack depends on the size of the occluded vessel.[1] In the

[1] Opinions vary as to the *cause* of the attack in embolism; Monakow and others assume a reflex cerebral anæmia.

occlusion of small vessels, there may be no attack at all. Frequently in the occlusion of a larger vessel, *epileptiform twitchings* are observed (disturbance in cortical blood supply). It is evident, that in a thrombosis that forms slowly, all phenomena of an attack may be absent.

But taken altogether, stroke phenomena extending over a long period of time afford a better prognosis in embolism than in cerebral hemorrhage. In the former case one may always hope for compensation from the collaterals, while in the latter, the severe attack is the result of a severe destruction of the brain. In the attack in embolism, the pressure phenomena, such as the initial sinking of the bodily temperature, the slowing of the pulse and the distant effects upon the medulla oblongata (disturbances in urination and in respiration) do not develop. The face does not show the intense flushing.

In respect to *focal signs*, we can, in vascular occlusion also, distinguish *transitory* and *permanent* signs. Precisely in embolism, *hemiplegia* and *hemianæsthesia* may totally disappear, in case the collateral circulation proves to be of sufficient compensatory power.

If a *softening focus* develops, there naturally appear *permanent* phenomena of functional loss. Concerning the local diagnosis, we refer to the preceding chapter. I should like to emphasize, furthermore, that just in embolism and thrombosis a *transient* hemiplegia may be followed by a second seizure with permanent hemiplegia.

Since the *left* a. fossæ Sylvii or its branches are most frequently occluded, *hemiplegia* of the right side with *aphasia* is the most common focal symptom (in left-handed people, softening foci of the *right* hemisphere may cause aphasia).

Diagnosis.—As has already been said, it is often impossible to distinguish between hemorrhage and vascular occlusion.

The differentiation of a thrombotic occlusion from a cerebral embolism or apoplexia sanguinea may be impossible. So great a diagnostician as *v. Bamberger* has said that he ventured but rarely to make a differential diagnosis in an apoplectic attack.

Besides the atheromatous process, the acute infectious diseases especially lead to thromboses (typhoid, diphtheria, in rare cases, pneumonia also). Recently I observed after pneumonia in an adolescent, a cortical motor aphasia (cf. *Port*, Münch. med. Wochenschr., 1909). Thromboses occur also in the puerperium. Changes in the vascular wall and in the blood may co-operate. Thromboses have also been observed after CO-poisoning and after narcosis. Infectious and toxic processes may also lead to the formation of thrombi in a weakened heart, and then embolisms may occur. In the infectious diseases, the embolism may also originate in an endocarditis. In senile (arterio-sclerotic) cerebral softening, *thrombotic* vascular occlusion is very common. As to *syphilitic* vascular changes (endarteritis obliterans) one must naturally take into account an exact anamnesis, and a thorough examination.

The question, whether *embolism or cerebral hemorrhage* is present, often can not—as has been stated—be answered at all. There is no certain scheme of differential diagnosis regarding this matter.

Hemorrhages occur preferably in older persons, embolisms at any age. But the main thing is to try to establish the *point of departure of an embolus.*

(*Valvular defect.* Ophthalmoscopic examination for *embolism of the art. centralis retinæ.*)

Treatment.—*In general*, the treatment is the same as that in hemorrhage. *Syphilitic processes* demand energetic *specific* treatment.

If the diagnosis "vascular occlusion" is probable and the *heart* is *weak*, then one should give camphor and digitalis. I have several times seen a striking effect from improvement in the heart's action; it is evident, that thereby the opening and blood supply of the collateral vessels must be improved.

(d) Syphilitic Affection of Cerebral Vessels

Syphilis causes an endarteritis obliterans in the cerebral vessels; frequently only isolated smaller sections of a vessel or of a branch of a vessel, are changed, while all the other portions of the vessel in question are normal. In contrast to arterio-sclerosis, there are often also *periarteritic* changes.

The *clinical* phenomena of *endarteritis syphilitica* are those of *vascular occlusion* (encephalomalacia). In every apoplexy, especially in the young, one should look carefully for signs of a past syphilis (anamnesis)! In a case, correctly recognized, energetic antisyphilitic treatment must be instituted at once. There is peculiar applicability here, of the following aphorism "*qui bene diagnoscit, bene medebitur.*"

In the syphilitic vascular diseases, there is usually no tendency to fatty degeneration and calcification, as in arterio-sclerosis, but, on the contrary, to *fibrous* transformation. Thus, occasionally in certain cases, a portion of the art. basilaris may be transformed into a solid fibrous cord. In others, aneurisms form in the basal cerebral arteries (*Kaufmann*). *Narrowing of the lumen and thrombosis lead to occlusion of the path of circulation.* To *Heubner* belongs the credit of being the first to recognize the clinical importance of syphilitic vascular disease of the brain. The basal vessels and the vascular region of the *a. fossæ Sylvii* are preferably affected.

(e) Cerebral Arterio-sclerosis as a Chronic Disease Picture

In the preceding chapters, we have discussed the clinical features of *apoplexia cerebri.* Cerebral hemorrhage, embolism and thrombosis may cause it. All three of these possibilities may develop upon a basis of athero-sclerosis.

We have discussed the various forms of the course of the attack. All the phenomena may be transient in light cases, in others, a number of symptoms of functional loss persist (*hemiplegia*).

Sometimes the strokes recur within a short time; besides the motor phenomena of functional loss a quickly increasing *dementia* may be noticeable.

But in some cases, there need be no accumulation of attacks; one light attack opens the unspeakably sad final scene of a life successful in its mental activity, sufficing to lead the owner of a highly developed brain to *dementia*.

Nay, every outspoken attack may be lacking and a *decrease in mental capacity* may present the main symptom of the *cerebral vascular disease*. "On a l'âge de ses artères," this aphorism holds good not only for arterio-sclerosis of the peripheral vessels and the arterio-sclerotic diseases of the heart, but for the *brain* as well. Arterio-sclerosis is a "*using up*" disease. How often we find precisely in mental workers, that their approaching old age is manifested above all, in a serious change in their psychic and intellectual qualities. We find that a man has grown old after we have transacted business with him, after we have heard him speak in public, when we find some change in his nature. We say, he has no longer the old elasticity of his mind; his speech is halting, his memory faulty, etc. Pressure on the head, headaches, are frequent complaints in arterio-sclerosis of the brain and they may precede the serious phenomena for years. It is evident that just in such cases, that is, in persons of great mental activity, the *differential diagnosis between so-called neurasthenic* complaints and *arterio-sclerosis* of the brain is not always easy, but, on the other hand, one must remember that the "using up" disease of arterio-sclerosis develops more easily in markedly neurasthenic individuals. Still there is a great difference, whether a patient has always been neurasthenic, or whether neurasthenic symptoms, which were formerly quite foreign to him, and for whose appearance there is no particular reason, have suddenly developed in *advanced age. Precisely the sudden and apparently unmotivated appearance of neurasthenic complaints must rouse a suspicion of cerebral arterio-sclerosis.*

The progressive decrease in functional capacity of the cerebral cortex may cause the patient to despair, and drive him to suicide. Psychically there appears an egocentric narrowing of the emotional life (*Ziehen*).

The aged man loses the understanding of and sympathy for his surroundings; association of ideas and productivity decrease.

A feeling of dizziness, of confusion, conditions of excitement alternating with depression, delusions, lack of orientation, temporary dysarthria and amnesia appear as consequences of a temporary disturbance in circulation or of a summation of small and very small softening foci (encephalomalacia multiplex). The picture of pseudo-bulbar paralysis may develop also. But genuine bulbar symptoms may appear also. It is characteristic of the arterio-sclerotic psychic disturbance, that in general it spares the "*nucleus* *of personality*" longer than, for instance, paresis. Credit belongs to *Cramer* and *Weber* of having shown in individual cases how much of the personal individuality is carried into the disease picture.

The anatomic picture consists mostly of thrombotic occlusions of small and even the smallest vessels with the resulting softening foci; besides there are atrophic processes.

Particularly distressing for those associating with such patients are the not uncommon *crying spells.* Laughing spells do not occur so often. Finally the picture of total senile dementia develops. But besides these disturbances of the *psyche* and the *intelligence*, there appear motor disturbances, that have nothing to do with hemiplegia. The patients exhibit a peculiar disturbance in gait which resembles *paralysis agitans* (pro- and retropulsion) or *basophobia:* uncertainty, tripping, slight wavering, and above all an extraordinary tendency to fatigue, are noticed.

The patient may have slept well (sleeplessness is frequently, but not always present); he wishes to take a short walk after breakfast. At first, everything goes relatively well. After a walk of fifteen minutes there is suddenly such uncertainty that he can go no farther. He must be led. Patients at this stage of the disease should not be allowed to go out alone. The impression is created that in this peculiar disturbance in gait, *organic and functional* disturbances co-operate. A patient, who understands his condition, experiences a new psychic shock therefrom daily. *Oppenheim* suggests as the cause of the disturbance in gait a disturbance of the centers for equilibrium and of the co-ordination apparatus. *Naunyn* sees in this disturbance more the result of the *general* cerebral degeneration.

Possibly the following history of a case (own observation) may portray the disease picture—which is as important as it is difficult to describe, in its peculiar development—better than an enumeration of all the separate symptoms.

Eminent physician, 65 years old, with large medical practice, has been complaining for two or three years about a slight feeling of numbness in his right hand and the frequent appearance of pressure and pain in the head. Especially during psychic excitement, marked tremor of the hands. He takes everything seriously; is a little suspicious. Professional duties, that had formerly not excited him in the least, now cause him anxiety and sleepless nights (he becomes more and more anxious; fears to exceed maximum doses; the giving of disease certificates leads to doubts and long discussions as to whether he is writing the certificate correctly, is making no mistakes, etc.).

Pronounced arterio-sclerosis of the palpable arteries.

One day, an *apoplectic attack* with slight temporary disturbance of consciousness. Hemiplegia of the right side, which in the course of about eight weeks disappears almost completely. Only slight paresis.

He again takes up his practice; can walk from one to two hours without becoming very tired; takes trips on the train.

As a result of the two months rest necessitated by the attack, he even appears considerably improved in physical and mental capacity. But he is frequently very much depressed. For about 6–8 months, he follows his vocation again. Very gradually, his memory becomes worse and worse. In the course of a year, such *uncertainty and fatigue in walking* develop, that the patient can no longer go out alone.

He often cries and speaks of suicide. Joyful occurrences in the family cause a temporary improvement. But finally the dementia increases more and more, the patient must be watched even in his room. He must be fed. He can no longer read. He can no longer light his cigar. Usually he gives himself up to quiet brooding, and suddenly begins to cry passionately. Conditions of violent excitement appear. Urine and feces are discharged into the bed.

Since nursing at home is becoming impossible, he is taken to the hospital.

There he *rests in bed for several weeks.* To the joy and astonishment of his family and those about him, *the patient recovers so far that he can walk alone with the help of a cane. He watches operations in the hospital with interest.* He reads again with interest; recites the classics; jokes; visits friends. In brief, he displays a mental activity such as has not been observed in him for 2–3 years.

This improvement persists for 1–2 months; then there appears a dementia, which progresses slowly, but with no further remissions.

Death after one year (3 years after the only apoplectic seizure; no more attacks occurred later).

General *motor weakness* and the described *disturbances* of the *intelligence* and the psyche dominated the disease picture.

This case shows at the same time the remarkable, surprising improvements, which may occur in the course of a general arterio-sclerosis of the brain, and which must be judged very cautiously, in so far as the prognosis is concerned.

The *pathological physiology* of so remarkable a disease picture has received clearer explanation by the more recent investigations on arterio-sclerosis and the vascular innervation of the brain.

Arterio-sclerosis leads to a decrease in the expansibility of the vessels. The flow of blood into the capillaries is made more difficult. *Jacob Henle* already taught us: "The movement of the blood is mainly dependent on the heart, the distribution of blood on the vessels." *Senac* said: "Les artéres sont de vrais coeurs sous une autre forme." If the vessels of an organ are arterio-sclerotically affected, this appears above all in a poor adaptation of the vascular system of the organ in activity. Arterio-sclerosis thus leads to a poorer supply of blood to an active organ. Decrease in work performance and tendency to fatigue are, therefore, necessary consequences of vascular deterioration.

Then because of vascular occlusion, there occur *disturbances in nutrition and injuries to tissues; the vessel disturbance is associated with smaller hemorrhages* (*encephalomalacia multiplex*) and *atrophic* processes.

Besides the anatomic and physical changes in the vascular wall, the injury to the vaso-motor activity brought about by the arterio-sclerosis is also of the greatest importance. Thus *Romberg and Otfried Müller* could show that arterio-sclerotic arteries react more slowly to stimulations of cold and heat than healthy arteries. In most recent times, *Müller* and *Siebeck* have cleared up the vaso-motors of the brain (cf. introduction to this chapter, p. 662 ff.). The fluctuations in this peculiar symptom complex must be traced back to fluctuations in the circulating blood supply of the organ.

It is not far fetched when one seeks and finds parallels in pathological occurrence between certain cerebral disturbances, intermittent claudication and coronary sclerosis.

I should like to emphasize one additional point: it is not impossible that at times the lighter uræmic phenomena are overlooked or incorrectly interpreted. (Slight stupor, headaches, malaise.) One must watch the heart (hypertrophy of the heart). Kidney disease can at once make itself noticed by an *increase* in the *blood pressure*, which precedes albuminuria and cylindruria (importance of measuring the blood pressure and exact examination of the urine).

The **treatment** of general arterio-sclerosis of the brain makes great demands on the physician. The disturbance of the psyche and the intellect demands much tact and patience. The general treatment is, on the whole, quite the same as that of arterio-sclerosis. Mental and physical rest, decrease in further deterioration. Corresponding diet: more vegetables, milk; less meat. No coffee. Little alcohol. Administration of iodides. Care as to regular evacuation of the bowels. In cardiac weakness, digitalis. Thereby the blood supply of the brain is increased. The dangers of giving digitalis in arterio-sclerosis have been much exaggerated. If the cardiac condition demands digitalis, it must be administered. With *high* blood pressure (contracted kidney) nitroglycerine should be used, but carefully.

In a high degree of dementia, or dangerous conditions of excitement, institutional care is essential.

(f) Meningeal Hemorrhages, Hæmatoma of the Dura Mater

Here we exclude all discussion of *cephalhæmatoma neonatorum* (through tearing of the subperiosteal vessels in difficult births). (Cf. text-books on surgery and obstetrics, especially also *Runge*, text-book of diseases of the newly born.) (Lehrbuch der Krankheiten der Neugeborenen.)

The *hæmatoma* of the *dura mater* is of particular clinical interest; it is usually the consequence of a pachymeningitis *interna hæmorrhagica.*

Ætiologically, above all, *dementia paralytica* and *dementia senilis*, but particularly *potatorum*, are concerned (anamnesis).

In very rare cases, extensive meningeal hemorrhages occur in *scurvy, pernicious anæmia, leucæmia, typhoid, smallpox, kidney disease.* According to *Virchow* in pachymeningitis hæmorrhagica it is a question of a fibrous inflammation, the result of toxæmic alteration of the dura (Melnikow). According to *Huguenin*, it is a question of hemorrhages from vessels, that have been changed by disease and are liable to rupture. If a larger hemorrhage results, it spreads the pachymeningitic membranes apart. The escaped blood often rests upon the brain like a cap, and may cause deep depressions in the surface of the brain (*Kaufmann*). The effusion of blood may be absorbed and then there may result the formation of large, multilocular *cysts.* (*Hygroma duræ matris, Virchow.*)

The **symptoms** are, in the main, those of cerebral pressure. In a slowly occurring hemorrhage they develop quite gradually. A strikingly morbid desire for sleep may appear. The pupils are usually contracted. If the central convolutions are compressed, monoplegias and epileptiform twitchings may appear. If the pressure affects one-half of the brain with special force, then a hemiplegia may appear; *in such a case, differential diagnosis from a typical apoplectic attack is impossible.*

In several cases, that were carefully observed, there was even, strange to say, present clinically, a paresis, even a marked *hemiplegia of the same side as the hæmatoma.* In such cases, the seat of the hæmatoma, even though known as a probability, can not be definitely determined. Indeed, trepanations have been even made on the wrong side (*Kaufmann, Enderlen*).

In this respect, the following case, observed by me, is instructive:

N. Gr. 62 years old. Working man.

Brought unconscious to the clinic June 2, 1908. The people who accompanied him, said that the patient had suffered much in recent times from headache. He slept badly, and was occasionally very much excited.

On the evening of June 1, he became unsteady. His people believed he was drunk, since he was a drinker. He was put to bed and immediately became totally unconscious.

Result of the Examination.—Absolutely unconscious, slightly rigid, face flushed. Left pupil larger than the right; corneal reflexes abolished.

The left eyeball somewhat protruding.

Stertorous breathing. The left cheek puffed out in breathing.

Pulse slow, 48–60.

The left arm shows no movement at all; the leg, but slight movement. Patellar reflexes diminished.

In the *left half of the body*, pain sensation apparently lowered.

June 3, exitus letalis.

The autopsy (Dr. Schultze) **showed:** Dolichocephalic, irregularly shaped cranium. Skull cap heavy (500 g.). A large quantity of blood follows incision into the dura of the *left* hemisphere. Inner surface of the dura covered with a brownish, reddish membrane, to the anterior part of which a brownish coagulum clings. Quantity of blood 100–200 c.cm. The surface of the brain appears flattened, and between the dura and the surface of the brain, there is a space as large as a child's palm 1.5 cm. in height.

The inner surface of the dura of the right hemisphere shows some bloody spots that can be easily wiped off. The convolutions on the surface of the right hemisphere are flattened. When the brain is taken out, dark fluid blood appears on the inner surface of the dura at the base, especially of the middle cranial cavity. Cranial base, no change; otherwise, the rest of the brain shows no change.

In such cases, the other hemisphere must be exposed to specially severe pressure against the cranial wall, or at the foramen magnum a marked bend might work so that the pyramidal tracts of the other hemisphere are injured. Similar observations have been made in some cases of hydrocephalus (cf., moreover, also Broadbent's hypothesis).

Though a certain importance must be attributed to the gradual or *paroxysmal-like* appearance of the cerebral symptoms, with corresponding anamnesis (potatorium), still one must confess that the diagnosis could be definitely made only in very few cases. (*Griesinger* 1862.)

Traumatic hemorrhages of the vessels of the cerebral membranes, above all, of the *A. meningea media,* need brief mention here. In spite of the fact that they belong exclusively to the domain of the surgeon, exact knowledge of them is of the greatest importance to the *practitioner* for purposes of differential diagnosis, and above all, with respect to therapeutics, since such cases very frequently do not come first into the hands of the surgeon, but into those of the practising physician; it is a matter of regret, that the typical picture of traumatic hæmatoma from the A. meningea media was formerly far too little known, a fact which is best illustrated by the poor results obtained in cases that were handed over *too late* to operative treatment: of 99 of *Bergmann's* cases, only 16 recovered.

The A. meningea media, the largest branch of the A. maxillaris int., passes outward between dura and bones, in the region of the temples, dividing into an anterior and a posterior branch. In traumatisms of the head, that lead either to fracture or infraction of the cranium, but even in those that cause no visible or tangible injury to the skull, the artery may be injured (partial tearing or total division), very frequently beneath the os temporale and parietale. This injury to the vessels takes place usually on the side of the lesion, but in rarer cases, may be found also on the opposite side, through the influence of contrecoup. The typical clinical picture is often the following: a man receives a knock or blow on his head, or falls upon it. At first no severe symptoms besides the phenomena of a "bump" and a light commotio cerebri appear; the patient recovers quickly, stands up, and walks home, often without assistance. There, after several hours, appear very gradually, at first light, then more severe *general* phenomena of brain pressure, vertigo, headache, slow pulse, vomiting, etc. Now, if the extravasation of blood increases, there appear nearly always, through its pressure on the cerebral cortex, phenomena of paralysis; they appear gradually as a rule, but often also increase very rapidly; most frequently, their character is that of a hemiplegia, with which are combined occasionally unilateral spasms.

If effectual aid is not given, the paralysis becomes more and more pronounced, the general pressure of the brain greater and greater and the patient perishes in a state of deep coma. The only treatment in cases like that described is naturally an operation, as soon as it is possible to make a diagnosis. And this can usually be made from the characteristic history and the course. The operation must be performed by opening of the cranium, if possible, not by trepanation, but by osteoplastic resection over the cortical parts, which, depending on the type of paralytic phenomena, must be supposed to be affected. As soon as the bone is removed, a large flat coagulated clot of blood protrudes immediately, after the removal of which is perceived an often surprisingly deep impression on the cortical region. The hemorrhage must then be arrested, according to the usual rules of surgical technic, by ligature and tying.

The **prognosis,** if the case has been operated upon *in time*, is not unfavorable. Often the paralytic signs begin to decrease within a few hours after the operation and may then disappear absolutely in the course of days or weeks. The prognosis becomes more unfavorable, of course, the later the patient is operated upon, that is, the longer the local and general effects of the brain pressure have persisted.

(g) Thrombosis of the Cerebral Sinuses

(*Sinus Thrombosis*)

We distinguish between a primary, so-called marantic, and a secondary, inflammatory thrombosis (thrombo-phlebitis). The inflammatory thrombosis is usually the result of a caries of the petrous bone (otitic sinus thrombosis). It leads to purulent leptomeningitis, extradural abscess and abscess of the brain, especially in the cerebellum.

Marantic thrombosis appears as the result of a severe general disease, which has caused cardiac weakness, injury to the blood (changes in coagulability) and injury to the inner wall of the sinus. Its *seat* is usually in the sinus *longitudinalis* and sinus *transversus.* The mechanical consequences of occlusion of the sinus are: congestion, edema, and hemorrhages into the brain. It may lead to extensive encephalomalacia (red softening).

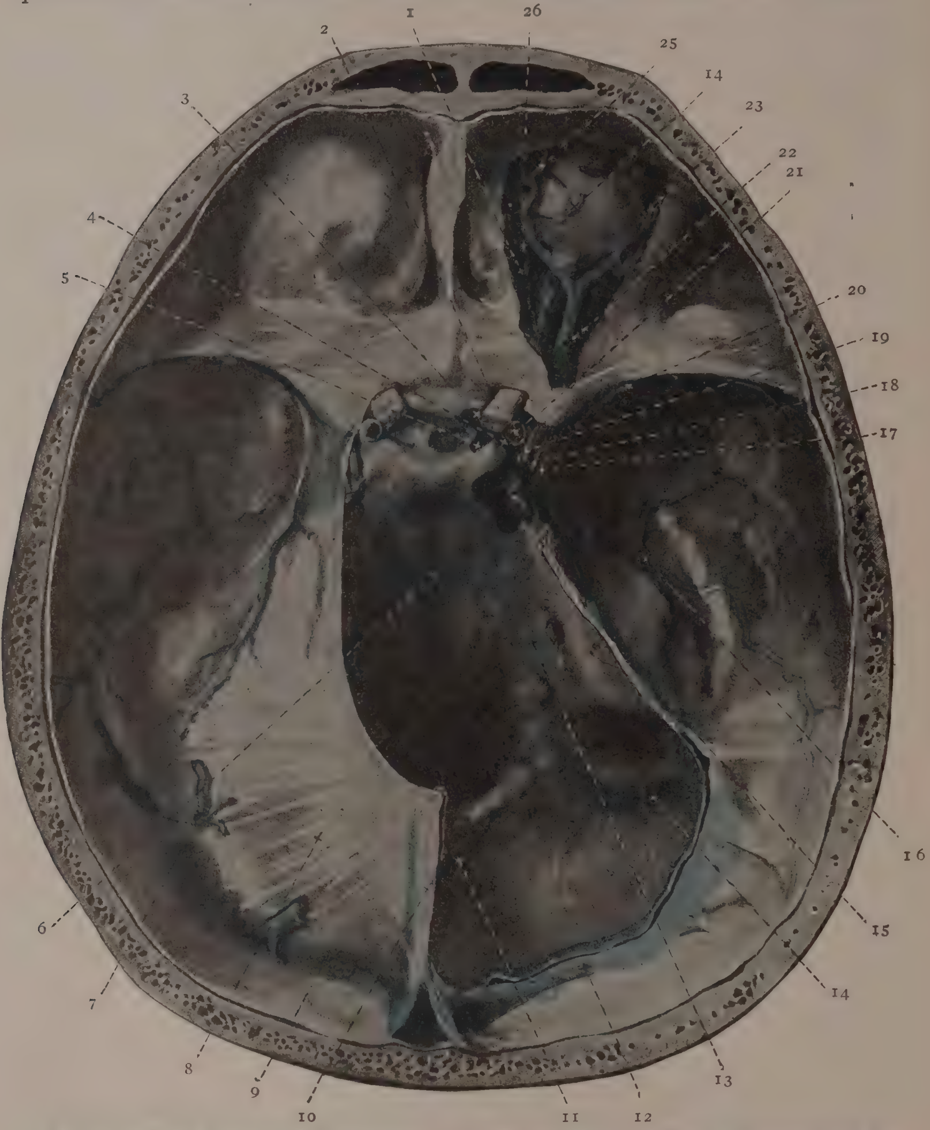

FIG. 183.—Sinus duræ matris (from above). (*From Spalteholz.*) At the right the orbit and the sinus cavernosus are opened and the tentorium cerebelli removed. 1, V. ethmoidalis post.; 2, N. opticus; 3, Sinus intercavernosus ant.; 4, Sinus cavernosus; 5, Sinus intercavernosus post.; 6, Plexus basilaris; 7, *V.* cerebri; 8, Tentorium cerebelli; 9, Sinus transversus; 10, Sinus rectus; 11, Sinus occipitalis; 12, Sinus transversus; 13, Sinus petrosus inf.; 14, Sinus transversus; 15, Sinus petrosus sup.; 16, VV. meningeal mediæ; 17, N. maxillaris; 18, N. troch.; 19, N. occulomotorius; 20, Sinus sphenoparietalis; 21, A. carotis interna; 22, V. ophthalmic sup.; 23, V. lacrimalis; 24, V. vorticosa; 25, V. nasofront.; 26, V. ethmoidalis ant.

If both sinus transversi are occluded, naturally the entire brain will suffer; whereas when only one is occluded, a certain compensation is always possible. In occlusion of the sinus longitudinalis, of course no compensation is possible.

If we recall (introduction) that a high degree of vascular stasis in the brain, enclosed in its rigid cranial cap, necessarily leads to increased difficulty and decrease in the arterial inflow, the severe disturbance of the functions of the central organ is intelligible. Apathy, sopor, coma, are frequently the most important *general* phenomena; paralyses, delirium, and spasms, are less common. Therefore it is not strange that anatomic casuistry usually designates "*meningitis*" as its clinical diagnosis. In some cases, however, the diagnosis was successfully made. In 1862, *Griesinger* diagnosed a thrombosis of the sinus transversus (edema behind the ear). In making a diagnosis, one must observe whether, for instance, the thrombosis of the sinus transversus extends to the vena jugularis interna, and whether congestive phenomena (edema) are visible in external veins communicating with the sinus.

Thrombosis of the *sinus transversus* may lead to edema behind the ear. The V. jug. ext. on the same side may be less filled than on the sound side. According to *Gerhardt*, this is explained by the fact that the V. jug. ext. empties its blood more quickly into the imperfectly filled V. jug. int., in case the thrombosis extends to the latter.

Thrombosis of the sinus longitudinalis may lead to engorgement of the veins that pass from the temporal to the parietal region. This is clearly visible in children. As a result of overfilling of the veins of the nose, the nose may bleed profusely. Cyanosis in the region of the V. facial. ant.

Thrombosis of the sinus cavernosus causes especially severe congestive phenomena. Since the V. ophthalmica flows into this sinus, enormous eyelid edema (also edema of the face because of the communication of the V. ophthalmica sup. with the V. facialis ant.), exophthalmos, congestive phenomena in the veins of the retina, edema of the disc may appear.

In *inflammatory* thrombosis, paralyses of the eye muscles and neuralgias (first branch of the tri-facial and ophthalmia neuroparalytica) are observed. (Vicinity of the sinus cavern. and the nn. oculomot. and trochlearis, first branch of the tri-facial and N. abducens.) Phlebitic thrombosis of the S. cavernosus is mostly induced by inflammations of the orbit and the face. The diagnosis of phlebitic thrombosis is supported by the proof of the presence of a primary inflammatory focus (ear, orbit, erysipelas of the face and head, traumas, caries, large boils), general pyæmic phenomena, septic *pulmonary embolism* (abscess of the lungs).

Treatment of phlebitic thrombosis must, above all, correspond to the indicatio causalis (cf. text-books of surgery and otiatry).

Marantic thrombosis demands before all, absolute rest. Treatment of the anæmia and weakness of the heart.

7. PROGRESSIVE PARALYSIS OF THE INSANE

BY

R. GAUPP (Tubingen)

Progressive paralysis of the insane is one of the gravest diseases of the central nervous system. Psychic disturbances, above all, an increasing mental deterioration, are combined in manifold ways with physical phenomena of paralysis, to form a varied disease picture; as a rule, the disease terminates in death within a few years. The anatomic foundation of the disease is usually a chronic, rarely an acute destructive process in the entire central nervous system, especially in the cerebral cortex. The nervous parenchyma (cells and fibres) are severely injured, in part entirely destroyed; at the same time, inflammatory phenomena appear in the blood-vessel system, which are also most pronounced in the cerebral cortex. Besides this, there is proliferation of the glia. At the basis of this morbid disease process, there is evidently a toxæmia, which can be brought into relation with the previously acquired syphilis. Progressive paralysis of the insane, and tabes are "metasyphilitic" nervous diseases. The disease seems to become more and more frequent with the growth of large cities; in Berlin, male paralytics form 30–40% of the male admissions; in country districts, the disease is comparatively rare, even to this day.

Synonyms.—In Germany: dementia paralytica, progressive paralysis of the insane, recently often "paralysis" for short. In France: paralysie générale progressive, maladie de Bayle. In England: paresis, paretic dementia. The name given it by the laity, "softening of the brain," is to be discarded as it is anatomically incorrect.

The disease is nowhere described in old medical literature; its exact description is not yet a century old. *Bayle* (1822) recognized first the connection between and the parallel development of the physical and psychic symptoms. In 1826, *Calmeil* was the first to treat of the disease in a monograph, tracing it back to a periencephalitis chronica diffusa. The more recent, more certain development of histopathology of the cerebral cortex furnished a secure pathologic anatomical foundation for progressive paralysis.

The definition of the concept "progressive paralysis" varies even to-day. In France, some authorities reckon as progressive paralysis many things differently named by us. With the separation of the arterio-sclerotic, alcoholic, traumatic, tertiary-syphilitic forms of dementia, the conception of a progressive paralysis becomes gradually narrower and clearer. The more successful this differential diagnostic separation was, the more evident became the regularity of the course. The older literature must therefore be used only with the greatest caution, relative to its discussions on prognosis, recoveries, and results of treatment. In spite of all diagnostic advances, there are still cases, in which the postmortem only furnished a sufficiently clear final explanation. We owe to *Nissl* and *Alzheimer* the certain diagnosis of progressive paralysis from the histopathological picture. The macroscopic findings in the cadaver do not always make possible a definite decision. Therefore the physician must be careful in expressing a positive opinion.

Causes and Occurrence of the Disease.—The importance of race and nationality is as yet under debate. Among many nations (Chinese, Turks, Irish), progressive paralysis is supposed to be rare, in others, as for instance, the Magyars, especially frequent. But it is doubtful whether this has anything to do with differences in race. Only in about 40–50% of the patients are psychic or nervous diseases found in the family, or habitual intoxication, or other signs of degeneration. Some paralytics have inherited a small brain; frequently other signs of degeneration are found in them. Men are far more commonly attacked than women; the proportion fluctuates between 7 : 1 and 2 : 1, in large cities, progressive paralysis of women is relatively more frequent than in the country. Most "paretics" are attacked between the 35th and the 50th years. Before the 28th and after the 55th year, progressive paralysis is rare, but occasionally it occurs even in the seventh decade. Only about 0.4% fall ill before the 25th year. In men the higher vocations are preponderantly affected (officers, manufacturers, merchants, officials, very rarely clergymen); in women, the lower ones. Waitresses are often attacked; the view formerly held that prostitutes are rarely "paretics" has proven to be an error.

The most important cause of progressive paralysis is preexisting *syphilis*, which was contracted at most 10–15 years before the outbreak of the disease. The experiences of recent times (careful statistics, results of comparative psychiatry, of lumbar puncture, serological investigations) make it more and more probable that *Möbius* was right in explaining progressive paralysis as a *metasyphilitic disease;* that is, the anatomic changes in progressive paralysis are not typical syphilitic tissue changes; this paralysis, therefore, is not a form of tertiary syphilis, although that person only can become a "paretic" who has formerly had syphilis. Constitutional syphilis produces some changes in the organism, probably chronic poisoning; the poison created leads to the progressive destruction of the central nervous system. Many other things also speak for the great importance of syphilis in the ætiology of progressive paralysis. Experiments proved that the progressive paralytics could not be infected with chancre virus. Because women usually develop venereal diseases at an earlier age than men, they develop progressive paralysis at a lower average age. The disease has been frequently observed in married people. Not rarely, one of the couple is a progressive paralytic, the other a tabetic. Progressive paralysis of children and adolescents arises on a basis of hereditary syphilis. Where syphilis prevails, there progressive paralysis is usually prevalent also (large cities; frequent occurrence among officers, merchants—rare occurrence among clergymen, educated women; no one ever yet heard of a nun with progressive paralysis). On the other hand, it is by no means proven that this form of paralysis is the consequence of a particularly severe attack of syphilis; one finds very often, on the contrary, that the former infection was accompanied only by very few symptoms. Nor does it seem to make much difference, whether the syphilis was carefully treated or not.

Not every syphilitic becomes a "paretic"; in fact, there are some countries with much syphilis and little progressive paralysis (Persia, Bosnia, etc.). The disease, therefore, needs still other factors to bring about its development. Many accuse the increased difficulty of the struggle for existence, the hurry and flurry of modern life, the greater consumption of nervous vitality in the large cities. *Krafft-Ebing* coined the much quoted phrase, "civilization and syphilization" as the cause of progressive paralysis. It may be that mental over-exertion and especially frequent illhumor may pave the way for the disease; but I have known many progressive paralytics, in whose anamnesis nothing of all this was found, who had lived quietly and care-free, until they were attacked by the disease.

Recently on the ground of experiences in foreign, especially Mohammedan countries, the importance of *chronic alcoholism* as an additional factor has again been emphasized. Chronic alcoholism alone does not cause progressive paralysis; alcoholic "pseudo-progressive" paralysis is absolutely different from the real paralysis. Nevertheless it is remarkable how often one hears that progressive paralytics have formerly been in the habit of drinking much; and among the Mohammedans, of all the former syphilitics, those mainly seem to be attacked by progressive paralysis, who have adopted west-European customs and are addicted to alcohol. It is doubtful whether *sexual excesses* play any causal part.

Severe *injuries to the head* may cause a mental weakness, which, to a certain extent, resembles progressive paralysis, but is usually not progressive (so-called traumatic dementia). It seems, also, that a serious traumatism in the head may evoke progressive paralysis in a former syphilitic, and aggravate one that already exists. But in practice, in the supposition of such a causal relation, great care should be used, since for various well-known reasons, there is a tendency to-day to assign accidents as causes of diseases. Furthermore, traumatism is sometimes the result, not the cause of progressive paralysis: the patient falls during the paretic syncope, or as a result of paralytic ataxia and inattention.

Pathological Anatomy.—In the cadavers of progressive paralytics, numerous changes are found, some of which occur in many chronic diseases of the nervous system, while others are characteristic in their grouping of progressive paralysis. To the first group belong: thickening of the skull cap with wasting of the diploe, adhesions of the dura to the skull cap, pachymeningitis, hydrocephalus externus and internus, clouding and thickening of the pia (especially over the frontal lobes, while the occipital lobes remain free), adhesions of the pia with the cortex cerebri, cortical peeling, ependymitis granularis, more or less marked atrophy, and decrease in the weight of the cerebrum and the cerebellum, especially of the frontal lobes (narrowing of the convolutions, deepening and gaping of the sulci), diminution of the thalamus opticus, reduction in size and atrophy of the entire spinal cord. Of other macroscopic changes frequently found we must mention arterio-sclerosis in the cardiac portion of the aorta, and in the larger vessels of the base of the brain, chronic atrophic and degenerative processes in the musculature of the heart, in the liver and the kidneys, general brittleness of the bones, due to osteoporosis.

All these findings are not pathognomonic for progressive paralysis; nor are they found in all cases. In the *histological* examination, the *pia* is always found to have been changed by the disease; there is infiltration with plasma cells, lymphocytes, and mast cells; the *vascular apparatus* exhibits proliferation and degenerative processes. The *cortex cerebri* itself contains, in every case, diseased blood-vessels; the endothelium is proliferated, and forms new vessels through sprouting and vascularization of the proliferated intima. The elastic tissue is increased, the adventitia proliferated, the adventitial lymphatic sheaths distended and infiltrated; plasms cells, lymphocytes and mast cells are always there found in them. In the further course of the disease, phenomena of involution appear in the vessels (obliteration of the vessels, hyaline degeneration). In the cortex are found *Nissl's* rod cells originating from the vessels. The ganglion cells of the brain are affected in various forms, and often break down rapidly; many perish gradually, and the cellular architectonics of the cortex is disturbed. The medullated fibres of the cerebral cortex disappear early and in large numbers. The supporting tissue, the glia, proliferates; masses of abnormally large gliar cells arise, forming many fibres; thus thick meshes of fibres develop in the white and gray matter. The gliar sheath of the vessels is thickened.

Not all parts of the brain are affected at the same time and with equal severity. The loss of the fibres begins first in the delicate structure of the 2d and 3d Meynert layer; the projection and subcortical association fibres are preserved longest; the upper layers of the cerebral cortex are, as a rule, affected before those situated deeper. The regressive process is usually most marked in the frontal brain, least in the occipital brain; the temporal lobe is now more, now less affected. Another localization may occur exceptionally (atypical progressive paralysis). The *cerebellum* is the seat of changes similar to those found in the cerebrum; often the *thalamus*, too, is affected; other parts of the brain also in slighter degree (pons, ventricular gray matter, medulla oblongata). The *spinal* cord is probably changed by the disease in all cases; in some cases degeneration of the columns predominates, in other it is a matter rather of diffuse degenerative-atrophic processes. Progressive paralytic disease of the posterior columns is not definitely distinguishable in anatomic respects from the tabetic; the degenerative processes in the spinal cord predominate over the inflammatory ones. Frequently the lateral columns are affected; still oftener there are found combined columnar diseases. In the gray substance of the spinal cord, also, degenerative changes appear occasionally. Changes are rare and of little practical importance in the peripheral nerves and in the muscles.

Psychic Symptoms.—The principal symptom is a gradually increasing mental weakness. The patient shows, usually quite gradually, a transformation of his entire personality for the worse. Frequently, the disease begins with a "nervous" introductory stage; the patient becomes irritable; there is a tendency to passionate outbursts of anger upon slight provocation; he becomes fatigued easily; he is overwhelmed by a hypochondriac mood of depression; he loses interest in his work. What he acquires mentally with the greatest effort, is at once lost, likewise self-control and self-judgment, tact and ability to criticise, comprehension of the tasks and duties in his vocation and family. The finest psychic processes are soon lost; the patient becomes ethically and æsthetically duller, his character deteriorates. Sometimes even at the beginning of the disease he loses sense for outer forms, becomes tactless, careless in bearing and clothes, rude in behavior. Energetic natures occasionally become weak and limp with the onset of progressive paralysis, are very easily influenced, can be "managed better" than before, sensitive patients become dull or brutal. If a certain degree of excitement be added, the patient becomes conspicuous through his words and actions; he no longer controls his impulses, indulges in "dirty" expressions, behaves sexually in an obscene manner, eats greedily, succumbs to alcoholism. If he is irritated, he flies into a towering rage, scolds, using vulgar expressions. In high spirits and feeling of improved condition of health, there is often a period of excessive and silly activity; the patient forms senseless plans, engages himself to a girl of low rank, makes ridiculous purchases, gives valuable things away to strangers. For some time, he can still do quite well the work to which he has been accustomed, whereas every new mental acquisition is difficult, or already impossible. The memory becomes uncertain, first for the more recent past, the power of receiving and retaining new impressions is gradually lost; the order of events, as to when they occurred, becomes faulty; delusions of memory appear. Mental possessions formerly acquired remain somewhat longer, but often mistakes in counting early occur. Gradually, the intelligence becomes dulled, attention uncertain, interest is lost, knowledge disappears. Temporal and local orientation are lost, the surroundings are no longer observed; the patient goes astray when he takes a walk.

The gradual deterioration of mental powers is usually not perceived at all; there is, as a rule, no psychic feeling of illness. Only in the beginning of the disease, the progressive paralytic sometimes has an undefined suspicion of the coming misfortune. At this time suicide is not uncommon. The critical faculty suffers more and more during the progress of the disease; the patient is evidently becoming demented. Gradually, a slight stupor is noticeable; the progressive paralytic often lives as if half asleep. The picture may now vary according to the emotional mood. Sometimes the mental breakdown is quite gradual; all psychic processes deteriorate steadily and slowly, until the patient finally lives a mere vegetative existence. In euphoric dispositions, on the other hand, we see excitements with ever-changing grandiose schemes, senseless actions, many grotesque ideas of grandeur of varying content; the patient revels in millions and billions, is emperor, god, super-god, etc.; the maniacally colored excitement may be increased to the confusion of a flight of ideas; in a mad frenzy, the patient jumps, dances, laughs, weeps, destroys; he soils things with urine and feces, tears his clothes, attacks those about him. His mood is suddenly transformed from one of pure joy to one of violent rage, during which the patient may become brutal and very dangerous. With the decrease in mad excitement, his megalomania sometimes disappears; but often it persists in spite of the appearance of superficial quiet, and in the progress of the dementia, the patient in a dull brooding manner, expresses ideas of exaggerated grandeur and persecution, that are perfectly nonsensical. Then, stupid euphoria usually dominates the disease picture up to the very end.

In other cases the mood is, for a long time, preponderantly despondent. Hypochondriac and melancholy hallucinations develop, which are frequently utterly absurd. Nihilistic dementia occurs especially among depressive "paretics." Actual delusions are less common. Loud dialogues, that the progressive paralytic carries on with himself often cause us to suspect hallucinations. In blind tabetic-paralytics, numerous optical hallucinations are often observed. Systematized ideas of persecution and catatonic symptoms are unusual in progressive paralysis, but occur as transitory, less frequently as permanent, psychotic pictures. Much confabulation is common. In the further progress of the disease, the mental nature of the patient becomes duller and duller, he becomes quite inactive, mumbles apathetically to himself in a condition of stupor; he has to be fed, does not recognize his relatives, may become entirely asymbolic. Cheerful, angry, anxious and hypochondriac moods may be entirely lacking, or appear in mad succession. Finally, the dementia does away with all mental life. The patient becomes childish, finally, almost like an animal.

Physical Symptoms.—*Headache* may be permanently absent, but is frequently present, especially early in the disease; often it is almost unbearable; it may resemble migraine. Occasionally one finds persistent *sleeplessness*, in other cases, great weariness and *somnolence.* Appetite and digestion may suffer, the appearance becomes unhealthy. The patient seems to age quickly, he becomes emaciated. The *expression of the face* changes, the face is more relaxed, vacant, the muscles of the face unilaterally paretic (flattening of the naso-labial fold); in laughing a flash-like twitching passes over the face; the muscles of the lips tremble, when the patient tries to speak. In the execution of commanded movements (mouth shut, eyes open—eyes shut, mouth open) the patient is awkward and clumsy. The tongue, when protruded, often swerves to one side; it frequently exhibits trembling or fibrillary twitchings. The timbre of the voice may change, the voice becomes more monotonous, harder, bleating; the formerly musical patient sings falsely. *Speech and writing* are affected in manifold ways. The disturbances in speech in progressive paralysis are as a rule a combination of cortical and bulbar phenomena of functional loss; at first the cortical loss predominates, later, the bulbar paralysis is dominant, finally aphasia and asymbolia rule supreme. Speech at the beginning is often slow, monotonous, stammering; trembling of the muscles used in speech is observed; occasionally hesitation and stumbling over syllables appear early, afterwards literal paraphasia. Later speech becomes indistinct, slurred, dragging. With increase of dementia, the formation of sentences and the verbal expression of thoughts suffer.

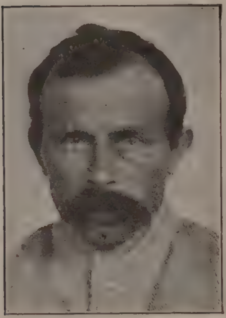

Fig. 184.—Repressive form in the early stage. Flaccid features.

Speech is tested in various ways. First one should observe the patient during spontaneous speech, when he is relating the story of his life, in a natural, unembarrassed manner; then one should set him certain tasks: repetition of difficult words, counting from 650 backwards to 620, reading aloud from a book. At the same time one should notice whether the patient reads intelligently and with expression. The more quiet and the less embarrassed he is, the more certainly the speech disturbance found may be regarded as an actual sign of the presence of the disease. After attacks and at a later stage in progressive paralysis, there are very often suddenly added to the speech disturbances already mentioned, aphasic, paraphasic, asymbolic disturbances; difficulty in *finding words* may appear early. Towards the end of the disease, the power of speech is sometimes almost or entirely lost; the speech thesaurus has disappeared, and only a few words can be brought forth, and those wholly unintelligible. In times of excitement, one hears occasionally confused, indistinct talking to himself and stammering.

The *cheirography* of patients, who were used to writing, often shows changes of diagnostic value. They are in part motor disturbances (indistinct, atactic writing, trembling script); in part, they are the expression of psychic weakness, especially of the disturbance in attention and in sense for form (omission of letters and syllables, repetitions of letters, syllables, and words, untidy appearance of the written paper, with blots and crooked lines). Agraphia also is not rare. In the test the writing before the patient was stricken should be compared with that written afterwards.

The decrease in muscular strength and the increased deterioration in muscular control, which have given the disease its name "progressive paralysis," gradually make themselves felt in many other voluntary and involuntary movements also. Gait becomes more clumsy, awkward, and heavy. Ataxia and paresis are combined. Total paralyses of individual muscles or muscular groups are rare, and occur mostly only temporarily after attacks; on the other hand, the innervation of all the muscles loses power and precision, and a general motor awkwardness and heaviness results, which may gradually reach such a point that the patient becomes almost immovable, and lies, a helpless form in bed. Often, there is violent general *trembling*. The *grinding of the teeth* belongs to a later stage in the disease; it is observed especially in the attacks and in the ultimate dementia. Bulbar symptoms (paralysis in deglutition and respiration) frequently cause the end.

Besides these general disturbances in voluntary movements, there are found, usually at an early stage, diagnostically important disturbances in the pupils, in the reflexes, in sensibility and in passive mobility of the limbs. These symptoms are variously grouped. Most important are the *disturbances in pupillary reaction*. This reaction may remain normal in progressive paralysis throughout the whole course of the disease; as a rule, however (80 to 90%), there are found—and indeed usually quite early—changes caused by the disease: considerable pupillary difference with retained, slowed, weakened or lost reaction to light; miosis or mydriasis, the former especially in tabo-paralysis; unilateral or bilateral reflex pupillary rigidity, absolute pupillary rigidity. Of these disturbances, unilateral or bilateral *reflex pupillary rigidity* or sluggish pupillary reaction is the most important, since it occurs almost exclusively in tabes and progressive paralysis. During the further course of the disease, sluggishness frequently passes over into absolute rigidity. Slight pupillary differences with good reaction to light are not very significant, since they may be found also in healthy people and especially in many nervous patients. Occasionally, the left or the right pupil is alternately more dilated; sometimes they are irregular in outline. Hippus is rare, occurring exceptionally unilaterally. The pupillary disturbance may precede by years the psychic symptoms. Since the demented "paretic" may create difficulties in the examination of his pupils, the test must be made with peculiar care and patience; in mental diseases progressive paralysis is often diagnosed, because the examiner as a result of technical errors, believes that he has found pupillary rigidity. In doubtful cases, the test should be made in a dark room. The test is still more accurate, when performed with one of the more recent devices for measuring the pupils. One must guard against the common error of concluding from the presence of reaction to light, that no progressive paralysis could be present. In the spastic forms (severe speech disturbance, stiffness of the limbs, exaggeration of the reflexes) the pupillary movements sometimes remain permanently unimpaired. Frequently the consensual reaction to light is lacking even early. The absence of "secondary light reaction" (the pupil of the eye exposed to light becomes still narrower, when light is thrown upon the other eye also) seems to be a particularly important early symptom of paralysis.

Other *paralyses of the muscles of the eye* are rare. They occur in the same manner as in tabes (transitory or permanent paralyses of one or more nerves of the eye muscles), and are most likely to be observed in tabo-paralysis. The same holds good for *blindness* through *atrophy of the optic nerve.*

Tendon Reflexes.—The changes in the patellar and Achilles tendon reflexes are important. Sometimes they are unequal, occasionally abolished, often weakened, more frequently increased; they rarely remain continuously normal. Genuine ankle clonus and patellar clonus occur, but are, on the whole, rare. The test must be made with the leg bare. In miosis and reflex pupillary rigidity, the loss of the patellar reflexes is quite common; in mydriasis and absolute pupillary rigidity, they are usually exaggerated; at the same time, there are present in such cases severe *spasms* (*hypertonia* of the muscles) and atactic paretic symptoms on the part of the limbs. But there are various exceptions to this rule. Occasionally, the tendon reflexes are at first active, even abnormally increased, only to be abolished later (combined columnar disease in the spinal cord).

Cutaneous Reflexes.—They are not rarely abolished. The *Babinski* sign is found exceptionally in the spastic forms; more frequently it appears unilaterally after paralytic attacks, which leave behind them hemipareses. The same is true of *Oppenheim's* leg reflex.

Sensitiveness of the Skin.—In tabo-paralysis, it may be disturbed as in tabes. In the other forms of progressive paralysis, there is, as a rule, a general hypalgesia traceable to the psychic condition.

The *senses of taste and smell* are often lowered considerably during the course of the disease.

The *Romberg sign* is sometimes found, but more often it is lacking. More severe ataxia of the legs with flaccid joints is usually absent, belongs chiefly (excluding final conditions) to progressive tabo-paralysis. Abnormal relaxation of the joints with increased tendon reflexes occurs, and speaks for a combined columnar disease in the spinal cord.

Peripheral paralyses (radialis, peroneus) and circumscribed muscular atrophies are rarities.

Loss of sexual power may appear early; sometimes it is preceded by a period of increased libido. *Bladder disturbance* is common. In the later stages, there is incontinentia urinæ and alvi.

The *paralytic attacks* are frequent and important symptoms of the disease. They vary clinically: brief attacks of vertigo, vaso-motor disturbances, attacks of migraine with amaurosis partialis fugax (Flimmerskotom), syncope with or without epileptiform twitchings, general or unilateral twitchings, or twitchings confined to one part of the body (face, arm, leg) with or without loss of consciousness. The twitchings are exceptionally synchronous with the pulse. They may last for days. In the beginning of the disease, syncope is common. After the attack, there are often aphasia and paraphasia, ptosis, hemiplegia or monoplegia, mostly of but short duration; also hemianopsia, paralysis of touch, and similar cortical phenomena of functional loss, both motor and sensory, are observed. The persistent presence of severe local phenomena (aphasia or hemiplegia) speaks in general against progressive paralysis, but occurs exceptionally; spastic contractures may then develop. During the attack, there is often fever up to 104°. The patient frequently dies during the attack. If he recovers, the symptoms of progressive paralysis, especially dementia and speech disturbances are more pronounced afterwards; sometimes retrograde amnesia persists for weeks and months. The simple demented form with severe spastic-paretic symptoms especially has many attacks. Where attacks have once appeared, they usually return and then preferably in the same form. This holds good particularly for atypical (Lissauer's) paralysis. The anatomic foundation for the attacks is seen in an acute increase of the paralytic process in certain cerebral regions, which is supposed to be caused by a sudden overflow of the suspected paralytic virus.

As *attacks of psychic nature* must be interpreted many quickly appearing and often quickly disappearing conditions of delirious excitement or anxious confusion. Occasionally there develops a condition like delirium tremens, even when the patient is not a drunkard. Furthermore, there are found sometimes in "paretics," who, as a result of their disease, have become addicted to strong drink, acute psychotic traits, which clearly display alcoholic coloring (delirious disorientation, emotional delusions, marked tremor); with abstinence in an institution, these traits rapidly disappear. The examination of the *cerebro-spinal fluid* (lumbar puncture) shows an increase in albumen and an abnormally large number of lymphocytes. The pressure in the cranial and spinal space is supposed to be frequently much increased. According to the more recent investigations, there are found in the spinal fluid of "paretics" specific-syphilitic antitoxins, an important proof for the metasyphilitic nature of progressive paralysis;

it seems that during the disease, the central nervous system for a long time actively produces anti-bodies.

So-called "*trophic*" disturbances belong rather to the late symptoms. Considerable fluctuations in the weight of the body (quick decrease in weight in spite of sufficient nourishment, enormous accumulation of fat), increasing brittleness of the bones, falling out of hair, hæmatoma in the ear after slight mechanical violence, vesicular desquamation of the skin, mal perforant, decubitus are to be mentioned. The last often develops very rapidly during an attack. *Cyanosis* and *edema* of different parts of the body are not infrequently observed. In women, menstruation very often ceases.

Disturbances in the *temperature of the body* occur even at other times than during the attacks. Later in the course of the disease, subnormal temperatures are common. A quick rise in temperature sometimes precedes the attack.

The "paretic's" *urine* often exhibits anomalies: intermittent albuminuria, peptonuria, glycosuria, polyuria without sugar. We have not as yet satisfactory investigations into the changes in the condition of the blood.

It is customary in progressive paralysis to distinguish various *clinical forms*, according to the psychic symptomatic pictures that predominate. The very acute form may bear the traits of *delirium acutum;* anxiously excited or morbidly happy confusion with rapidly increasing megalomania may develop into wild cries, striking out with hands and feet, stereotyped running hither and thither; death occasionally results after a few weeks in utter exhaustion, not rarely after suppuration, lung diseases or other complications have set in. In this *galloping* form, attacks frequently occur; if the first violent onset is survived, there frequently occur remissions that last for several months and which may be so pronounced that one begins to doubt the diagnosis, especially if the physical symptoms are but little pronounced. More frequent is the *expansive* form, in which after a nervous or hypochondriac introductory stage, a disease picture like mania appears. Exalted moods, motor excitement, prolific scheming, many rapidly changing grandiose ideas, senseless actions, often sudden violent outbursts of rage, are here combined with paralytic symptoms and with weakness in judgment and memory. After the fading of the excitement, megalomania remains, and the symptoms of functional loss become more prominent. Less common is the change into a mood of depression, or even a constant change between maniacally and depressively colored conditions (so-called *circular* form of progressive paralysis). The agitated and expansive form of progressive paralysis combines preferably with a disease of the posterior columns of the spinal cord. Especially peculiar is the megalomanic confabulation of blind (opticus atrophy) "paretics" with pronounced tabetic symptoms. Somewhat less common than the expansive form is the *depressive* form, which often rapidly runs its course. Fantastic micromania, nihilistic ideas, mad hypochondriacal delusions, which may occasionally arise from an insane misinterpretation of tabetic pains and paræsthesias, ideas of persecution, startling conditions of anxiety, in which the patient can not be influenced, serious suicidal attempts, refusal to eat, sometimes dull brooding, are observed. Feeding with a tube may become necessary. Paralytic attacks occur, but are not common. The depressive form of paralysis is found with comparative frequency in women. Its course is rather rapid.

The most frequent picture, under which progressive paralysis nowadays appears, is simple chronic progressive dementia, sometimes beginning with a hypochondriac, later mostly with a euphoric mood, with few or no delusions (so-called *simple demented form of progressive paralyis*). In this form, attacks are especially common; the physical paralytic symptoms attain their highest degree, speech becomes absolutely unintelligible, general motor spastic paresis often causes utter helplessness. Finally, an asymbolic condition develops, which must be understood as a summation of many phenomena of functional loss. In these cases, the spinal cord usually exhibits combined columnar degenerations; occasionally only the lateral columns, more rarely only the posterior columns are affected. Death frequently results during a paralytic attack.

One may speak of a *paranoid* form of progressive paralysis, if ideas of persecution and delusions have a prominent place in the disease. This symptom-complex is usually present for a short time only in progressive paralysis and then passes over directly into euphoric dementia. Where systematized ideas of persecution and numerous delusions persist for a long time, one must be very careful in making a diagnosis of progressive paralysis. One should look for alcoholic, or syphilitic pseudo-progressive paralysis. Dementia præcox is also often mistaken for progressive paralysis.

The early (*infantile, juvenile*) *form of paralysis* occurs with approximately the same frequency in boys and girls, a fact easily understood, inasmuch as syphilis, congenital or acquired in childhood, is the main cause. In rare cases, the disease begins even before the 10th year; the majority occur between the 13th and the 18th year. The duration of the disease is longer than in the progressive paralysis of adults (4–7 years). In several cases, both father and mother were "paretics"; other transmitted taints were found in about half the patients. Some of the youthful "paretics" were mentally deficient from the first. Clinically, the simple form with dementia preponderates; numerous delusions of an expansive or hypochondriac sort occur, but are rare. Remissions of any duration are lacking, inasmuch as the course is chronic from the beginning. Paralytic attacks occur very frequently. The spastic forms with severe speech disturbance, general serious motor paresis, frequent attacks, are more common than the tabetic forms; in the latter, optic atrophy is usually found early. The patients are mostly behind in physical development. The course is continuously progressive towards death in extreme emaciation and general paralysis. In differential diagnosis, are concerned first of all, multiple sclerosis, diffuse gliosis of the cerebrum and tumor of the brain. The type of dementia, the pupillary disturbances, the speech disturbance, the character of the attacks, above all the result of lumbar puncture will, as a rule, make diagnosis possible.

The late form of progressive paralysis (*senile paresis*), in which the first signs are observed only after the 60th year, runs its course usually without stormy symptoms, without severe speech disturbance, without severe spastic-paralytic phenomena. Attacks are rare.

Tabo-paresis.—To pronounced tabes progressive paralysis is sometimes added later. Such cases often present certain peculiarities; the dementia bears other traits, the memory remains good for a longer time, speech disturbances and other motor disturbances may be entirely lacking, attacks are rare, the degeneration in character is more pronounced than the enfeeblement of the intellect, the patient remains psychically more active. The course is very slow; often no advance in psychic symptoms can be perceived for years; surprising remissions in progressive paralysis appear, as soon as the symptoms of tabes (ataxia, spasms) progress rapidly. The process of degeneration in the brain then shows an atypical localization. Not every tabetic psychosis is a progressive paralysis. On the other hand, there are also cases of tabo-paresis which display nothing unusual either in course or symptoms. One must distinguish from tabo-paresis, *the ordinary paralysis with predominant affection of the posterior columns.* The tabetic symptoms here usually remain few in number (pupillary disturbances, loss of the patellar reflexes, flaccid limbs, slight atrophy); marked degrees of ataxia are scarcely ever observed, perhaps because the patient dies before the disease is sufficiently advanced to produce severe tabetic symptoms. This tabetic form of progressive paralysis has, on the average, a somewhat longer duration than the spastic form, the speech disturbance is not so pronounced, attacks are rarer. But numerous exceptions to this rule are observed.

Combination of genuine progressive *paralysis* with *other diseases* (multiple sclerosis, syringomyelia, tumor of the brain, gumma cerebri) occurs, but is rare. In occasional cases, *epileptics* later have become "paretics."

Diagnosis.—Progressive paralysis is often at first mistaken for something else; at a later stage, the diagnosis is usually easy. The combination of organo-neurological symptoms with psychic phenomena of irritation and functional loss characterizes the disease. If a man between the 35th and the 50th year for the first time becomes physically affected, or without any real reason very "nervous," the physician must always think at first of progressive paralysis. Early symptoms important for diagnosis are: headaches, sleeplessness, *pupillary disturbances, loss of tendon reflexes* in the legs, considerable *exaggeration of these reflexes*, apoplectiform and epileptiform *attacks* with only transitory paralyses, temporary loss of speech, attacks of vertigo, increase of the lymphocytes and the albumen in the spinal fluid, specific syphilitic antitoxins in it, slight ataxia of the limbs, relaxed vacant features, paresis of the facialis, deviation of the tongue, marked tremor of the tongue, and *speech disturbance*. The disturbance in speech is the *most important* physical symptom of progressive paralysis, but often appears only after the pupillary and tendon reflex disturbances. In men, loss of sexual power is sometimes a striking early symptom; less common are optic atrophy, lancinating pains, and other tabetic symptoms.

Of early psychic symptoms, the following are especially important: change in character, increased irritability, quick change of mood, loss of the more delicate psychic feelings, mental heaviness, a willingness to be guided, diminution of the power to receive and retain new impressions, weakened judgment, lack of insight into the deterioration of the mental powers, unfounded euphoria, insane actions. To these are often added sooner or later, psychotic pictures of an expansive, hypochondriac, agitated, delirious nature.

Various tasks are before us in the *differential diagnosis*, at the various stages of the disease. In the prodromal stage are concerned: *neurasthenia* and the *psychopathic* conditions, with their increased exhaustibility, sleeplessness, irritability, the hypochondriac complaints, pains in the back and the head, nervous speech, trembling of the hands and the tongue, the pupillary difference which is occasionally present. In these conditions, however, all organo-neurological conditions, the entire change in character, the dementia are lacking; the memory by test proves to be objectively better than the hypochondriac patient confesses, the power of judgment has not suffered; the feeling of illness exaggerates the actual lack of physical and mental capacity. Patients who come to the physician because they believe they are becoming insane or demented, are usually not "paretics." Exceptions of course occur. Sudden changes of mood speak for progressive paralysis, obsessions against it. It must not be forgotten that a tabetic may be psychopathic or become neurasthenic. The psychic condition must be decisive. Expansive progressive paralysis may resemble *mania*, depressive progressive paralysis, *melancholia*. The synchronous presence of physical symptoms and the anamnesis (nervous prodromal stage with psychic weakness and attacks) usually protects us from errors. On the other hand, I should warn against concluding upon progressive paralysis from the unbounded megalomania, or the hypochondriac or melancholy hallucinations. Often, too, it is absolutely impossible to test intelligence and memory exactly in manic or depressed patients. An inhibited maniac (in so-called mixed conditions) may create the impression of a euphoric dement. The content of the hallucination has but slight diagnostic importance. One must guard against the confusion of speech mannerisms in manic or catatonic patients, with the speech disturbance in progressive paralysis. If the mania is combined with alcoholism, the diagnosis at a given moment may be particularly difficult.

Alcoholic dementia may, as long as the patient is under the influence of alcohol, resemble progressive paralysis (alcoholic pseudo-paralysis) very closely. Common to both diseases may be: tremor, ataxia, loss of the tendon reflexes, diminished pupillary reaction, paresis of the facialis, disturbances in sensation, trembling speech, flash-like twitching during speech, disturbances in writing, attacks, dementia, euphoria, irritability, brutalization of feeling, changing hallucinations, conditions of delirium. Alcoholic dementia, however, does not progress, if the misuse of alcohol is discontinued; furthermore, the reflex pupillary rigidity is lacking; the reaction to light, which is often weakened, is improved by abstinence from alcohol. The tendon reflexes, also, often return, because their loss depends upon a curable neuritis. The power to receive and retain new impressions is usually much poorer in alcoholic-polyneuritic psychosis than in progressive paralysis, the confabulations have more the character of delirious happenings; megalomania is found only rarely in times of acute, usually not long lasting, excitement. Lumbar puncture, as a rule, shows nothing abnormal in alcoholism.

The differentiation of progressive paralysis from other *poisonings* (lead, carbon monoxide, ergotin, morphine, opium, bromine, trional, bisulphide of carbon) may also cause difficulties *temporarily*. The same is true of the psychic symptoms of decay in uræmia and severe diabetes.

The simple form of dementia paralytica, when appearing in older persons, may be confused with *senile* and *presenile* dementia. In these, too, there is a weakening of the pupillary reaction with miosis, very rarely even complete pupillary rigidity. On the other hand, there are lacking, as a rule, in senile dementia: the speech disturbance, loss of the tendon reflexes, severe *general* dementia, and change in character, the all-around uncleanliness. The psychotic conditions of seniles (anxious excitements, deliriums) are usually aggravated towards evening, while the similar pictures in progressive paralysis do not show this dependence on the time of day. In senile psychosis, lumbar puncture shows normal conditions.

Arterio-sclerotic dementia may, especially in combination with senile cortical disease, create a disease picture like that of progressive paralysis. Attacks with more or less severe paralytic phenomena, emotional weakness, difficulty in speech, impairment of memory and of the power to receive and retain new impressions, are found in both diseases. The arterio-sclerotic dement, however, usually has a clear feeling of illness; he suffers from his disturbances; his dementia is not so general; his appearance less clumsy. The pupillary reaction is retained. The paralytic phenomena are usually from the beginning more severe and lasting (aphasia, hemiplegia, monoplegia). Arterio-sclerotics, also, are usually older than "paretics." Lumbar puncture, as a rule, gives normal findings.

The greatest difficulties in differential diagnosis are caused by *diffuse syphilis of the brain.* But here, too, the dementia is usually not so general nor so severe as in progressive paralysis; the critical faculty suffers less; memory remains intact for single events; the external condition of the patient is better preserved; often there is stupor rather than real dementia; disturbances in speech and writing are very seldom pronounced. Megalomania, delirium, attacks occur in both diseases. The course of cerebral syphilis is more changeable, not necessarily progressive; mercury and iodides help often, though not always; frequently, severe permanent paralytic symptoms appear (paralyses of the eye muscles, mono- and hemiplegias). The spinal fluid in cerebral syphilis contains, as a rule, less albumen than in progressive paralysis. The *combination* of syphilis of the brain and progressive paralysis is very rare.

Confusion of paresis with *multiple sclerosis* is usually prevented by considering the difference in age, the entirely different course, the different character of the disturbance in speech, the different condition of sensibility, of tremor, the absence of severe psychic phenomena of decay in sclerosis. On the other hand, the differential diagnosis between diffuse gliosis of the brain and infantile progressive paralysis may cause exceptional difficulties; but these cases, because of their rarity, are practically rather unimportant.

Tumor of the brain and progressive paralysis may resemble each other in the beginning. In the former, the headaches are more severe and stubborn, frequently localized; and there often exists the choked disc, that is lacking in progressive paralysis; marked stupor that lasts a long while, circumscribed headache, slow pulse, vomiting, persistent paralytic phenomena speak for tumor of the brain; general dementia, reflex pupillary rigidity, stumbling over syllables for paralysis. A confusion of progressive paralysis with *epilepsy* can probably always be avoided by the study of the entire anamnesis and with careful physical examination.

Course and prognosis.—Various stages of the disease are usually distinguished in progressive paralysis. In the *prodromal stage* (stadium prodromale) there are present undefined nervous symptoms, which make differentiation from neurasthenia impossible, or, at any rate, make it very difficult. This stage may last for months or years. After this comes the stadium *initiale,* in which the beginning of the psychic breakdown, and some physical symptoms can be found by careful examination, while the patient does not, as yet, seem to the laity mentally ill, but only nervous, excited, aged. This period, too, may last for months and even years, before it passes over into the stadium "*acme*," in which all paralytic symptoms are pronounced, and which usually necessitates treatment in an institution. The end is the stadium *terminale*, in which the patient, now an idiot, pines away helplessly and, with gradual decay of physical and mental powers, approaches dissolution. Death results, as a rule, in 2–4 years after the beginning of the first certain symptoms. A longer duration (4–10 years) is occasionally observed. Many patients die during a paralytic attack, others perish of somatic disease (pneumonia, suppuration, pyelitis, tuberculosis, etc.), a smaller number die in marasmus paralyticus.

Numerous exceptions to the course sketched above occur. Temporary improvements (remissions) are observed. But the disease is incurable, and, as its name indicates, progressive. Death is the regular outcome. No treatment can change this. Of course, individual cases of recovery are reported in the literature, but probably mostly, if not always, the cases were wrongly diagnosed. It has also been frequently reported, that the disease remained stationary. The investigation of this question proved to me, that it was probably always a matter, not of progressive paralysis, but only of symptomatically similar diseases.

Total and partial remissions are to be separated; the former are very rare; some physical symptoms, like reflex pupillary rigidity, can not retrogress. As a rule, during remission, the psychotic symptoms (megalomania, anomalies of mood, excitement), disappear; in the more favorable cases, the speech disturbance also; where this persists, the improvement is not of long duration.

Remissions are the more frequent, the more acutely the psychotic picture appears; they are almost entirely lacking in the simple demented form. Following acute febrile diseases surprising improvements have often been seen even at a late stage, and for some time the disease may come to a standstill. The therapeutic attempt to improve the disease by artificial suppurations can show individual cases of success, but can not yet be regarded as an unobjectionable method of treatment, inasmuch as there are in it dangers for the life of the patient. In any case, it does not secure permanent recovery.

Treatment.—Prophylaxis is more effective than any treatment: the spread of sexual diseases should be prevented. If a patient has contracted syphilis, he is, naturally, to be treated carefully with anti-syphilitic remedies; his life should be one of abstemiousness and he should be temperate both in work and in pleasure. As soon as the diagnosis, progressive paralysis, is established, mental and physical rest are to be ordered. Mercury and, especially, iodide of sodium may be tried at the beginning of the disease, but only when the general condition is good; usually, there is no result. If the patient is but poorly nourished, anti-syphilitic treatment should not be attempted. It must be tried, however, when the question has not been definitely answered, whether progressive paralysis or diffuse syphilis of the brain is present. Intravenous injection of salvarsan may be used. (*Burr*).

"Paretics" are usually not fit subjects for public sanatoria. Treatment by cold water, travelling, exciting pleasures are injurious. Use of alcohol should be forbidden, the use of tobacco restricted. In emaciated patients, an increase in weight is desirable. For severe headaches, cold compresses should be tried first. If hypnotics are required, one should at once give medium sized doses, since experience has shown that small ones are of no avail (veronal 0.6–1.0; paraldehyde 4–6 grams, trional 1.0–2.0). In conditions of sudden excitement, the only relief is afforded usually by hyoscin (subcutaneously, 0.001–0.002 pro dosi); besides, rest in bed or the continuous bath. Lumbar puncture is of no avail, therapeutically. In paralytic attacks one should see to the evacuation of the intestines and bladder, give chloral or amylhydrate by enema; besides this, an ice-bag to the head, water or air cushions to lie upon, infusion of salt solution.

The irritable and constantly busy "paretic" belongs in a private institution, so that he may not injure his reputation by insane actions, madly squander his fortune, become a tragic and horrible sight to his children and those about him. Nor should he have further chance to beget children, since the children of "paretics" are very frequently degenerates. The demented patient suffers little or not at all from compulsory confinement. With increase of the dementia and the physical decay, the demands for care become so great, that they can generally be met only in an insane asylum. With refusal to eat and attempts at suicide, this is absolutely necessary. An early appointment of a guardian on the ground of mental disease, may prevent much misfortune. It is the duty of the physician to point this out from the first to the relatives of the patient; often it is only attended to after the patient has caused some mischief.

Medico-legal.—The increasing mental weakness leads, especially with simultaneous expansive excitement, to minor offenses: verbal insults, resisting an officer, assault and battery, statutory offenses occur occasionally at the beginning of progressive paralysis. More common are drunken brawls, obvious, clumsy thefts. Every "paretic" is irresponsible (penal code 51). Very early in the illness he can no longer attend to his business (civil law code, 6), must therefore be disqualified, especially if he lives outside of an institution. The decision in difficult judicial questions (validity of a testament drawn up at the beginning of progressive paralysis, of a marriage concluded after the outbreak of the disease, cessation of disqualification during a remission, etc.) are best left to the alienist.

8. EPILEPSY

BY

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The current conception of *epilepsy* (falling illness, "Fallsucht," morbus sacer) dates from a time, in which one was accustomed in pathology, to consider the symptoms as the diseases. Whoever suffered from attacks, that were accompanied by unconsciousness, was considered to be an epileptic. Further experience taught, that so-called epileptic convulsions occur in various and different diseases, and that, on the other hand, there is an epilepsy without convulsive attacks. Thus in the course of time, the conception "epilepsy" had varying definitions, and even to-day, it is by no means certain where its boundary is to be fixed. It was proven, first of all, by the teachings of brain pathology, that epileptic convulsions of a general or circumscribed nature might be a symptom of a general or circumscribed cerebral disease. In progressive paralysis, multiple sclerosis, tumor and abscess of the brain, in cerebral syphilis, meningitis, poisoning by alcohol, lead, morphine, in uræmia, diabetes, myxedema, epileptic attacks may occur which, in purely symptomatic respect, can not be distinguished from the attacks of the disease "*genuine* epilepsy" ("*idiopathic* epilepsy"). The inevitable conclusion from this is that the conception of epilepsy as a separate disease must either be given up, or that one must look for other characteristic symptoms of *epilepsy* beside the attacks. Thus, the psychic disturbances and the permanent symptoms acquired recently greater nosological importance. Therefore we must not be surprised that the recent advances in our knowledge of epilepsy have come chiefly from psychiatry: the mental peculiarity of the epileptic, the slight periodic psychic anomalies are more and more used in the diagnosis of genuine epilepsy. At the same time, new problems have arisen also for ætiological and pathologic-anatomical investigation, after it was proved that one can not always tell from the disease picture whether "genuine" or "symptomatic" epilepsy is concerned.

With the group of diseases designated as *epilepsy* in the following presentation, we do *not* include these: the epileptic or epileptoid attacks in acute and chronic poisonings from metallic poisons (lead, arsenic), from alcohol, morphine, and in chronic mental diseases, such as dementia paralytica, dementia præcox, in circumscribed diseases of the brain, such as cerebral tumor, cerebral abscess, multiple sclerosis, cerebral syphilis, cysticercus cerebri, traumatic softening of the brain or cicatricial changes in the separate parts of the brain. No more do the attacks in cerebral infantile paralysis (encephalitis), porencephalia, meningitis belong to genuine epilepsy. On the other hand, it is neither practical, nor always scientifically possible to decide whether epileptic symptoms that appear in association with dementia, are to be considered as genuine epilepsy, or as symptomatic convulsions of a not specifically epileptic cerebral disease. Similar difficulties may arise also in the so-called traumatic epilepsy; doubtless, there are cases, in which a traumatism caused no demonstrable *circumscribed* injuries to the brain, or the cerebral membranes, but only *diffuse* changes, which clinically with other symptoms evoke epileptic attacks, or transform an epileptic predisposition into pronounced epilepsy. The relations between alcoholism and epilepsy are separately discussed later (p. 722).

By the disease concept "epilepsy" as it shall be described here, *we understand a chronic, usually progressive disease of the brain, the main symptom of which is a disturbance of consciousness, appearing suddenly and in the form of attacks.* Motor and other irritation phenomena are common, but by no means present in all cases. Besides the *transitory attack symptoms*, which occur often, *but not always periodically*, there appears in the majority of cases a *gradual transformation of the entire psychic being*, which sometimes chiefly affects the character, in other cases the intelligence as well of the patient (*epileptic degeneration*) and in the severe forms of the disease ends in terminal *dementia* of a high degree and peculiar coloring.

The disease is by no means rare; there are about 35,000 epileptics in Germany.

Symptomatology.—The *classical attack* (epilepsia major, haut mal).

Prodromal symptoms.—Occasionally the attack sets in without any prodromal phenomena; the patient falls over unconscious, after just having expressed himself as feeling perfectly well. In other cases, especially when the attacks are less frequent, but set in with violence and in rapid succession, certain *prodromal symptoms* precede the attack. (With *Binswanger*, we distinguish the more distant "heralds" from the real "aura".) Such prodromes, which may be noticeable a few days before the attack, are above all, changes in mood and in feelings. The patient becomes irritable, melancholy; he is oppressed by gloomy thoughts, feels depressed, his head is heavy, tired, he does not want to work; sometimes there is a migraine-like headache; the patient has abdominal disturbances, abnormal hunger or total absence of appetite, his sense organs are supersensitive. Vaso-motor disturbances may appear (urticaria, erythema, edema). Less common than the moods of depression is an unmotivated euphoria and state of joyful excitement. Such prodromal symptoms may appear in a patient with such regularity, that his relatives may know from his behavior, when an attack is imminent.

Aura.—By aura we mean the phenomena that immediately precede the convulsive attack; they last only a few seconds, at most, a minute. In the majority of cases of genuine epilepsy, all aura is lacking; it is most common in the forms in which a circumscribed cerebral disease or injury preceded the outbreak of epilepsy. The clinical pictures, under which the aura appears are varied, but in the epileptic, if an aura is present at all, the same form is likely to occur over again. By *psychic* aura one means a sudden change in the psychic condition: anxious excitement, the appearance of unmotivated fear, beaming happiness, the feeling of being dazed, as if thought were suddenly at a standstill, confusion and flight of ideas, ending in total loss of consciousness, arbitrary recurrence of certain memories in clear vision, peculiar "clear view" of former happenings, the feeling of having lived through the present situation before, painful distinctness of a single idea. The psychic aura passes over directly into total unconsciousness. After the attack, there is sometimes amnesia of the aura; in other cases, memory of it is retained (cf. later twilight conditions). A *sensory* aura may appear in very different forms: occasionally, there are elementary sensations of light or sound (seeing sparks, hearing a rushing noise, or a loud report), sometimes there are obvious hallucinations (figures, landscapes, above all, red, always very gay images): A bitter or metallic taste, a strange smell may precede the attack. By *sensible* auras one designates paræsthesias, pains in individual limbs, headache, abnormal visceral sensations. A *motor* aura is found mainly in symptomatic epilepsy as a result of organic cerebral disease. To this aura belong localized clonic and tonic spasms, which precede loss of consciousness, and are an important indication for the seat of the cerebral disease. On the other hand, certain complicated movements of a voluntary character appear also in genuine epilepsy, as, for instance, movements of stepping and running, (*aura cursativa*), unbuttoning of the vest, etc. The variety of such forms of motor aura is great. Spasms in the realm of the respiratory musculature are observed also (singultus, spasmodic sneezing or yawning, etc.) and speech disturbances (aphasia, stammering). We may designate by *vaso-motor* aura, a paling or reddening, palpitation of the heart with increase in temperature, severe outbreaks of perspiration before the loss of consciousness. Frequently the aural symptoms described combine to form complicated pictures.

The attack itself.—After the appearance of an aura, or more frequently without such appearance, the real attack comes on suddenly. Consciousness is lost, the patient collapses, often injuring himself in the fall. At once all the muscles of the body are seized in a tonic convulsion; the shrill cry that many epileptics emit at the beginning of the attack, arises through tonic convulsion of the respiratory muscles. The head is forced backwards or to one side, the eyes, wide-open, stare into empty space or are in extreme lateral position, the jaws are tightly locked. As a rule, there is opisthotonos. The arms are extended and rotated inwards, the hand clenched into a fist, the thumb pressed into the palm of the hand. The legs are usually in a position of forced extension. Breathing is at a standstill. All the muscles feel as hard as boards; no passive movements can be performed by the examiner. This tonic convulsion lasts usually from 15–30 seconds. Then the patient gradually relaxes, and now clonic convulsions set in, occasionally preceded by a general tremor in the limbs. The movements are not always of the same character; in general, there is in the limbs an interchange of movements of flexion and extension; head and trunk are hurled hither and thither with great violence, the clonus of the respiratory muscles creates panting and gurgling noises. The face is distorted by wild grimaces, the eyes roll convulsively, the tongue is thrust between the teeth. In this stage, injuries are of common occurrence. The contraction of the muscles of the bladder, the intestines and the sexual organs often lead to an involuntary discharge of urine, feces, semen. There is profuse perspiration over the entire body. The clonic stage lasts longer than the tonic (1–15 minutes); the violence of the twitchings gradually subsides, the intervals become longer, sometimes a severe tremor shakes the limbs, finally the entire body is again at rest; the loss of consciousness usually lasts a few minutes longer. Occasionally the patient awakes without the least suspicion of what has happened. In other cases, the patient falls into a sleep, which lasts for hours; often, there is only a certain difficulty in throwing off the stupor, which state gradually passes into the normal condition of consciousness. When the patient has recovered, he lacks, as a rule, all memory of the attack, sometimes also of the time immediately preceding. In some cases a feeling of perfect health returns very soon. More frequently there are certain distressing after-effects: enervation and weariness, headaches, unnatural moods, muscular pains, rarely vomiting. A feeling of being very well occurs also. *Very frequently* there are found upon very careful examination *transitory paralytic symptoms* both motor and sensory (aphasic and asymbolic disturbances, paralyses of the eye muscles, hemiparesis, blindness, deafness, narrowing of the visual field, hypæsthesias, and hypalgesias, of varying extent and severity.

At the beginning of the epileptic seizure, the face of the patient usually becomes pale, then after a short time, dark red, cyanotic. The veins are prominent in the neck, standing out like tensely filled pipes. The eye-balls become prominent; frequently there are small hemorrhages into the conjunctiva of the eye and into the skin (face, neck, breast). A point of predilection for such ecchymoses is the skin behind the ear (above the mastoid process).

The condition of the *reflexes* needs special consideration. The *pupils* in nearly all cases do not react during the severity of the attack; at first they are nearly always contracted, but soon dilate very much. After the attack has passed off there are occasionally oscillating movements of the iris. During the attack, *the cutaneous reflexes* are lost; but like the tendon reflexes, they usually are not to be tested with any degree of certainty during the attack itself. The knee jerks are often absent for some time after the attack; in the interim between the attacks, they are usually increased in epileptics. During and following severe attacks, ankle clonus and the *Babinski* sign occur. The temperature of the body is not as a rule elevated, but there are slight upward fluctuations immediately before and during the attack, which it is customary to consider as due to cerebral irritation. In the status epilepticus (cf. below) the temperature of the body rises several degrees.

Immediately after the convulsive attack, the urine often contains some albumen, rarely also acetone; the quantity of urine is occasionally increased. The urine, as a rule, contains much uric acid, the amount of which before the attack may have been abnormally low. *Krainsky's* theory that in epilepsy there is a periodic formation of ammonium carbonate, which causes the attacks and during them disintegrates into urea and water, is contested. The problem as to the chemical processes in epileptic explosions is not yet cleared up. Exact investigations are being carried on. The same holds true of the study of the toxic properties of the epileptic's blood (*Ceni* and others).

If the attacks follow one another in such quick succession that a new convulsion sets in before the preceding one is entirely over, the so-called "*status epilepticus*" arises, during which loss of consciousness persists. In such conditions death may occur from cardiac paralysis, superinduced by exhaustion. The status epilepticus, therefore, always directly threatens the life of the patient. The cause of such series of attacks is not known; but experience teaches, that if during the disease the attacks have been entirely wanting for some time, the appearance of the status is especially likely to occur unless the patient's condition is merely a light form of epilepsy. There are also cases in which the convulsions appear very rarely, but then always a number of them together. The supposition, that it is especially considerable disturbances in digestion which give rise to the status epilepticus, has not yet been definitely proved. All the residuary symptoms described above (paralyses, speech disturbances, stupor) are found frequently after the status epilepticus.

In some cases, the epileptic attack may be suppressed after the outbreak of the aura. If the aura consists in paræsthesias or twitchings in one limb, quick constricting or pulling of the limb will occasionally prevent the attack (cf. later under "reflex epilepsy"). Some epileptics, too, are supposed to be able by energetic exertion of the will, to retain their consciousness and thus to suppress the impending convulsive attack (*Oppenheim, Pick, and others*). No such case has happened in my experience. Other measures, too, (swallowing a little ordinary salt, calling out a certain word) are supposed to be able sometimes to prevent the outbreak of a convulsive attack.

Other forms of the epileptic attack.—Besides the typical convulsive attack with its tonic and clonic stage, there are in epilepsy numerous other forms of attacks, in which the convulsive element may be entirely absent or restricted to a few brief twitchings; often, too, attacks in *one* and the same patient are of different kinds. In genuine epilepsy the twitchings are rarely unilateral; sometimes, partial tonic and clonic convulsions exist side by side. Brief *attacks of dizziness* are very common; every thing swims before the patent's eyes, he has to hold on to something to keep from falling; at the same time, slight twitchings, discharge of urine, and other symptoms of a deep-seated disturbance in consciousness may appear. Occasionally there are only momentary gaps in consciousness, which are not perceived by the patient himself, and may even escape the notice of those around him. The patient interrupts his occupation for a moment, only to resume it immediately as if nothing had happened. Or he stops for a brief period while speaking, possibly continuing in the middle of a sentence, when the slight attack ("absence," petit mal) has passed over. These light attacks, which are very often accompanied by slight clonic twitchings, (movements of smacking and tasting, twitching in the limbs), may appear in a patient repeatedly every day. By epileptic *morbid sleepiness* (narcolepsia) one designates the peculiar attacks of suddenly falling asleep during the day, with consequent amnesis for the period directly preceding the sleep. Periodic *outbreaks of perspiration* with clouded consciousness occur as epileptic equivalents; *acute local edemata* are described also.

A rare form of epileptic attack is the so-called epilepsia *cursoria* or *procursiva*. Here, in the attack, a patient automatically performs movements of walking or running, which are absolutely unmotivated and accompanied with considerable dulling of consciousness; after the attack, there is no recollection of it. Sometimes it ends in general convulsions. Epilepsia procursiva is, undoubtedly, a severe form of epileptic disease, in which the serious permanent symptoms of the disease (degeneration, dementia) are probably never absent.

Of great practical importance is the fact, that the epileptic attacks frequently occur only *at night* during sleep (epilepsia nocturna) and may then remain unsuspected for a long time, if the patient is accustomed to sleep in a room by himself. Occasionally, he observes, the next day, a fresh bite in his tongue, or other injuries, a feeling of great weariness and psychic depression overwhelms him, he finds the bed clothes moist from the urinary discharge during sleep, and realizes from these evidences, that an attack must have taken place during the night.

Sometimes the attacks appear only at quite definite times of the day, or at definite occasions, for instance, during eating.

The transitory mental disturbances of epileptics.—"*Twilight*" *conditions* are frequent symptoms of epilepsy; in the majority of cases, they appear in connection with one or several convulsive attacks. Occasionally they precede an attack, and finally they sometimes replace the epileptic attack. Therefore, these distinctions have been made: *post-epileptic* (more correctly post-convulsive), and *pre-epileptic* (more correctly pre-convulsive) twilight conditions and finally, twilight conditions as *epileptic equivalents*. This distinction is from a practical standpoint rather unimportant; but the fact must be remembered that twilight conditions appear especially often after accumulated attacks. In rare cases, a convulsive attack appears during the course of the twilight condition. If such a condition occurs *before* an attack, the patient seems to be sometimes in a *depressed*, anxious mood, as if *weighed* *down.* He runs restlessly hither and thither, does not observe his surroundings, seems clearly stupefied, occasionally has headaches, dizziness, ringing in his ears, sees flashes of light. Some delusions may appear. This condition often, after an obvious aura, passes over into the real attack. Epileptic twilight conditions usually begin suddenly and also end rather suddenly. Consciousness is severely impaired, orientation seriously disturbed, the power of attention lowered, the personality to a certain extent dissociated from its normal connections. After the disappearance of the psychosis, there usually remains total or partial *forgetfulness* of the entire time during which the twilight condition persisted, often, also, for the hours or days preceding the psychosis, more rarely for a longer period (retrograde amnesia). Sometimes a long deep sleep puts an end to the twilight conditions. When the twilight conditions are of *longer* duration (they may last exceptionally for months, sometimes for weeks, more frequently for days or hours only), the disturbance of consciousness is occasionally less severe, the patient is apparently in his right senses, capable of sensible and complicated actions, and does not appear to those around him as a man mentally deranged. Occasionally he takes long journeys without any purpose, awaking at a strange place without knowing how he got there (poriomania).

The clinical picture of the twilight conditions is extraordinarily varied; usually the separate psychoses in one and the same patient resemble one another; this similarity may extend to the smallest details (duration of the twilight conditions, beginning and sequence of the symptoms, character of the hallucinations, delusions, compulsory actions). The predominant emotional tone is that of anxiety; less frequently, an ecstatic joy or a deep stupor inhibiting all emotional expressions may be observed. Catalepsy and instinctive negativism occur. Thinking is, as a rule, seriously impaired, orientation is lost, those surrounding him are misjudged according to his emotional tone. The verbal utterances are sometimes incoherent, only very exceptionally devoid of meaning; the severe associative disturbance is betrayed in the frequent appearances of echolalia, in perseveration, and in the combination of a confused state with aphasic and paraphasic symptoms. Very often, there is difficulty in finding words. In severe disturbance of consciousness, there may appear real asymbolia and apraxia. If there is pronounced dream-like stupor, numerous delusions usually prevail, which are chiefly of terrifying or ecstatic character. Sometimes the twilight condition assumes the form of *sleep walking.* It has always been emphasized that the hallucinations of epileptics are very frequently elementary; bright colors, especially red, roaring noises, fiery forms, smoke, lakes of blood, a pronounced foul odor are often perceived. *Religious* scenes are enacted: God, Christ, angels and devils appear, heavenly music sounds, the judgment day is portrayed before the eyes of the dreamily entranced patient. The occasionally horrible conditions of anxiety combine with instinctive excitement, lead to horrible deeds of violence, so that one may call the epileptic in the twilight condition the most dangerous of all sufferers from mental aberration. The motor condition varies very much; sometimes *stupor* persists throughout the entire duration of the twilight condition, in other cases, we see an alternation of stupor with excitement; occasionally the picture of an anxious *delirium* appears, which is distinguished from alcoholic delirium by the deeper disturbances in consciousness, the predominantly anxious emotional tone, the horrible delusions, the changes in consciousness of the personality (grandiose ideas), the more extensive amnesia. Sometimes there are threatening and scolding voices, hallucinations in which a certain systematization may appear, which urge the patient to impulsive deeds, and which cause fits of rage. Such deliria may last for weeks. If in such twilight condition the patient drinks spirituous liquors, the disturbance in consciousness is quickly increased, and the danger of passionate fits of rage and brutal deeds of violence grows (pathological intoxication). The amnesia for these periods is then almost total. Frequently the terrified patient ends his life; in other cases, the same criminal impulses recur with every twilight condition (immoral offenses, attempts at murder, self-mutilations).

Exceptionally, an epileptic twilight condition with predominantly cheerful mood and a flighty presentation of ideas devoid of any sense, may display an almost "manic" picture (so-called epileptic mania); but as a rule, depressed and anxious, sometimes, too, wrathful moods prevail; an alternation of depressed and expansive moods is rare. Of physical symptoms observed in the epileptic twilight condition, we must mention: analgesia of the body, narrowing of the visual field, dilated pupils in exceptional cases without reaction to light, assymetries in the facialis realm, increase of the tendon reflexes, albuminuria, slowing of the pulse (stupor) and acceleration of the pulse (excitement), increased secretion of sweat. Gait is sometimes stumbling, speech thick, monotonous or even mumbling.

In the more severe forms of epilepsy (early beginning of the disease, many attacks) twilight conditions are common; they are found, according to the computations of *C. Neisser*, in about 60% of the cases, in which the epileptics ought to be confined to an institution. Occasionally they appear, as do the attacks, at the time of the menses. As a rule, but not always, they appear only after epilepsy has existed for some time. Sometimes a long period free from attacks precedes their outbreak.

Other transitory psychoses in epilepsy.—From the real twilight conditions we must distinguish the very frequent *periodic fits of depression*, in which consciousness is but very slightly impaired, and after which memory is to a certain degree retained. Such depressed moods occur in about 75% of all epileptics; quite frequently they appear before the attacks; they may be so typical, as to make the diagnosis of epilepsy possible by their very occurrence.

They appear without external reasons from internal causes, therefore seem absolutely unmotivated, and as a rule last only some hours or days. Often the patient wakes in the morning in the irritated or anxious mood; everything angers him, an inner restlessness torments him, melancholy recollections haunt him, an undefined impulse drives him from work, he wanders about; the soldier deserts because a sudden painful longing for home seizes him. Sometimes a *hypochondriac* mood dominates him, or an *acute paranoid* symptom-complex with delusions; *cheerful* excitement with a peculiar *feeling of happiness* is less frequent. Usually there are combined with these endogenous morbid moods, and resembling the twilight conditions in this respect, transitory physical disturbances: palpitation of the heart, acceleration of the pulse, reddened face, marked perspiration, tremor, dilated pupils, seeing of sparks, headache, feeling of stupor. Alcohol increases this pathological state very rapidly, sometimes into a veritable twilight condition; if this has the character of pathological irritability, deeds of violence are often perpetrated under the influence of alcohol.

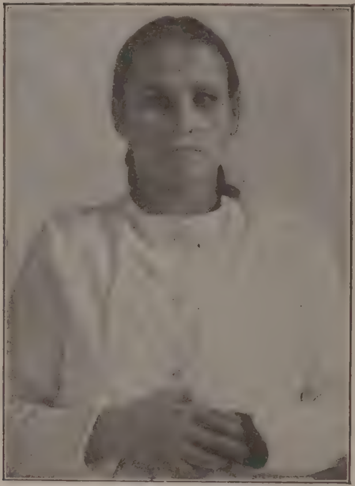

FIG. 185.—Young girl in epileptic mood (depression, inner tension, irritability, severe bursts of rage).

Finally, more recent investigations (*Kræpelin, Aschaffenburg, Gaupp*) have taught us, that real *dipsomania* is a form of epilepsy. By dipsomania we mean a *periodically recurring pathological impulse to drink, arising in an endogenous depressive mood of the patient.* The primary state is always the depressive mood, usually of the nature of anxiety and internal restlessness, often combined with a distressing feeling of thirst. This unnatural psychic condition drives the patient, though he may not care for spirituous liquors, to an insanely excessive use of alcohol, often continued for days, during which the excitement increases steadily, without the appearance of the physical paralytic symptoms of intoxication. Usually, the dipsomanic attack under the influence of the alcohol gradually passes into a severe twilight condition, from which the patient awakes with total or partial amnesia. If the patient is prevented from drinking during the attack (rest in bed, continuous watching, bromides, veronal), the morbid mood usually passes over quickly. Sometimes the dipsomanic is changed secondarily into a chronic drunkard.

From this genuine dipsomania are to be distinguished: the periodic excesses of chronic drunkards, who after vain attempts to be abstinent or temperate, fall under the influence of alcohol into severe acute alcoholism, also the excessive use of drink at the beginning of periodic mania and the attempts of psychopathic persons, who seek rest and forgetfulness in alcohol, to deaden their senses; their dysphoria, however, seems to have a psychological basis. The differentiation of dipsomania from hereditary alcoholism is more difficult, since in the latter also there is an inclination towards periodic excesses, and occasionally epileptoid traits appear. If we consider that epilepsy appears by preference in the children of drunkards, we shall understand that inherited alcoholism is apt to assume the epileptoid character of the periodic excesses.

The chronic change of personality in the epileptic.—In some light cases of epilepsy (rare attacks, late beginning, sober life) the disease may be restricted to the occasional appearance of attacks, while the personality on the whole remains intact. Some prominent men were epileptics. But far more frequently, in more than 75% of the patients, we see that the disease is of ominous importance for the intelligence and the character of the patient (*epileptic degeneration*). Of course, not all the striking peculiarities in the patient are to be interpreted without investigation as epileptic symptoms. Epilepsy grows in the soil of degeneration, and some epileptics have evinced isolated psychic stigmata of degeneration (irritability, egoism, intolerance to alcohol, etc.), before the first symptoms of the disease itself (depression, attacks) appeared. Besides this, it should be noted, that very many *idiots* have epileptic attacks (encephalitis, porencephalia, meningitis, etc.); the idiocy here, though it may resemble the epileptic variety, is not itself epileptic. But usually we see in genuine epilepsy, after it has been present for years, a specific transformation of the psychic being, peculiar to this disease alone. His character changes for the worse. The patient becomes irritable, stubborn, spiteful, quarrelsome, pedantic, petty, untractable, shameless, a liar, inclined to uniformly hypochondriac ideas, to fawning politeness and loquacity, becomes gradually more and more egotistic. He revels in praising himself and his family in bombastic terms, in boasting of his experiences and achievements. To this are added the signs of progressive dementia. Memory suffers, the power of receiving and retaining new impressions decreases, the power of comprehension becomes uncertain and very slow; there is weakness in judgment, the patient evinces a peculiar slowness, ceremoniousness, mental backwardness, and dearth of thoughts. Certain trains of thought recur incessantly. The movements of expression are exaggerated, the emotional impulses insist upon strong motor discharge; the sexual instinct frequently assumes a morbid direction; the mental interests disappear; the fondness for a sickly piety, which is often found in uneducated epileptics, finds expression in peculiar unmistakable outbursts of religious gush, the content of which is particularly poor. Slight causes occasionally produce insane bursts of rage. There is often remarkable optimism in the patient's judgment of his own disease. With the progress of the affection, the dementia may attain a very high degree; there may be excessive poverty of ideas, loss of the simplest conceptions, indeed even of speech itself and the understanding of speech. The patient becomes unclean, dull, can no longer occupy himself, lives occasionally in a perpetual twilight condition, in which the comprehension of the external world is made entirely impossible, while strong motor excitement finds vent in wild cries, running around, obsessional striking and destruction. Such patients, who naturally can be treated only in well-ordered institutions for epileptics, are extremely dangerous to those around them.

Epileptic dementia and degeneration of character are incurable. Of course, if through suitable treatment the number and severity of the attacks are lessened, a certain recuperation in the dull, apathetic, mentally thick-witted patient is occasionally seen. But here it is more a matter of getting rid of the symptoms of long-lasting twilight conditions, in which it is often difficult to decide just how far the patient has already been permanently injured mentally. The earlier epilepsy sets in, the more frequently the attacks occur, and the more psychic disturbances (equivalents, delirium, twilight conditions) are noticeable, the more severe the epileptic degeneration usually becomes. It has not yet been completely cleared up upon what it depends, whether an epileptic becomes idiotic or remains mentally active. Only quite recently, *Binswanger and Heilbronner* have emphasized that some epileptics, in spite of severe attacks, which have persisted for a long time, remain free from epileptic degeneration. Complicating alcoholism seems to influence more the appearance of severe twilight conditions, than the degree of mental decay. Frequent petit mal attacks have long been known to be especially destructive; with them, the psychic personality scarcely ever remains intact.

A singular form of mental disturbance in epilepsy is a *chronic-paranoic disease*, which may develop in mentally deficient epileptics from the acute paranoic excitements during temporary depressions; the hallucinations originating in the acute phase, are no longer corrected; there comes to be a permanent false belief in the sense of a persecution mania. Such a development is very rare; usually the hallucinations remain purely episodic disturbances.

Permanent *physical* disturbances are by no means regularly present in epilepsy; except for their attacks, the patients may appear entirely well physically. Of somatic symptoms there are found most frequently, disturbances in sensation (general analgesia, anæsthesias or hypæsthesias in spots). Sometimes the strength of the muscles seems slight in comparison to the development of the musculature. Very many epileptics are left handed. Often speech becomes slower, heavier, halting, in advanced dementia stammering. Scars, hemorrhages are consequences, not symptoms of the disease. (For the often numerous signs of degeneration cf. below p. 726.) Sometimes epilepsy is combined with myoclonus.

Causes.—If we understand by epilepsy, as has been presented above, only the so-called genuine idiopathic epilepsy, little that is definite can be said about the causes of the disease, while the *exciting causes* of the *individual attacks* are manifold.

Perhaps it is worth while to *give here a short summary of the conditions, under which we may see epileptic attacks occur without the presence of epilepsy itself.* The general convulsive attack occurs as a symptom of many general and local diseases of the brain: in progressive paralysis, dementia præcox, multiple sclerosis, tumor and abscess of the brain, cerebral syphilis, in acute and chronic poisonings (carbonic oxide, alcohol, morphine, lead, arsenic) in diabetes, nephritis, rachitis and cretinism, in arterio-sclerosis of the cerebral vessels, after severe concussions of the brain, in acute cerebral anæmia, through compression of the carotids (attempt at hanging). If there are scars in the brain itself or in the cerebral membranes (encephalitis, porencephalia, consequences of injuries to the cranium, the cerebral membranes, and the brain) epileptoid attacks may take place from time to time, which often, by the manner of their spreading (for instance, Jacksonian cortical epilepsy) indicate that they are symptoms of *local* cerebral disease.

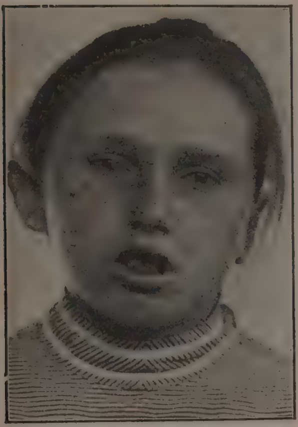

FIG. 186.—Epileptic girl, 11 years old. Extremely asymmetric cranium. Numerous signs of degeneration. Many attacks. Idiocy.

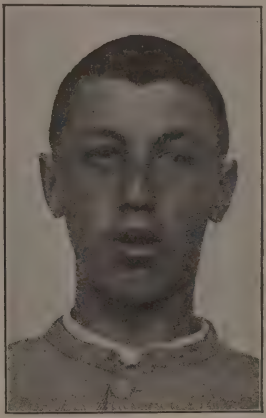

FIG. 187.—Twenty-years-old patient. Habitus epilepticus.

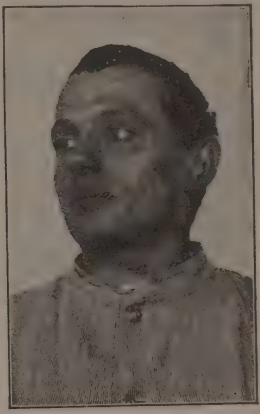

FIG. 188.—Twenty-seven-years-old patient. Epileptic character. Asymmetric cranium. Strabismus. Facial expression slightly dull.

The most important cause of *genuine epilepsy* is degeneration. The abnormal predisposition of the brain, upon which the disease arises, is inborn; in what it consists is not as yet known. Epileptics mostly spring from families in which nervous or mental diseases are common. Often the father or mother have also been epileptic; more frequently, there are found in the ancestors other nervous diseases, especially migraine. It seems, that epilepsy represents a more severe form of degeneration, which arises preferably when there is converging hereditary predisposition. Especially fateful is injury to the germ, due to the alcoholism of one of the parents. This is evident from many careful investigations. The assertion that drunkenness of the father at the moment of procreation causes epilepsy in the child, is naturally difficult to prove. The same is true of a number of other suppositions: advanced age of the parents, psychic shocks to the mother during pregnancy, injuries to the child's head during birth, are given as causes of the epileptic disposition. Tuberculosis, gout, chronic metallic poisoning (lead, arsenic) of one of the parents are supposed to be able to create in the children the predisposition to epilepsy (so-called "toxicopathic hereditary predisposition"). Consanguinity between psychopathic parents also is supposed to be a cause. If syphilis of the parents is inherited by the child, epileptic attacks may appear; they, however, are not as a rule to be reckoned as belonging to genuine epilepsy, but are rather to be conceived as symptoms of hereditary cerebral syphilis.

It is evident from two groups of facts, to what extent epilepsy represents a *form of degeneration:* the children of epileptics are almost always afflicted with one or the other of the nervous diseases or they are mentally abnormal; epilepsy of the parents lays a particularly heavy psychopathic burden upon the descendants. Furthermore, epileptics very often exhibit the physical and mental *stigmata of degeneration.* Many are nervous, psychopathic, from early childhood, before even the disease shows itself; many are born weak-minded; signs of physical degeneration (abnormal skull, hydrocephalus, microcephalus, asymetries of the cranium and face, abnormally high or flat palate, broad, heavy nose, thick lips, anomalies of the eyes requiring refraction, dilated, often differently sized pupils, squinting, abnormal formations of the ears, anomalies of the teeth, of the growth of hair, melanoderma) are found frequently in epileptics. More recent investigations (*Thiemich and others*) have taught us, that the *eclampsia* of infants is radically different from epilepsy; on the other hand, one often hears that epileptics, even in childhood, exhibited a tendency to spasms, suffered from pavor nocturnus, and by their obstinacy and bad temper, caused their parents much trouble in having them educated. In other cases, however, the disease strikes quite suddenly and unexpectedly persons who have heretofore been well and apparently normal.

The relations of *chronic alcoholism* to epilepsy need special discussion. We have already spoken of the importance of drunkenness in the parents. In pronounced epilepsy, alcohol, as has been remarked, undoubtedly aggravates the number and severity of the attacks, favors the appearance of serious psychic disturbances, makes the epileptic violent and dangerous. In other cases, the impression is given that an epileptic predisposition was transformed only through alcoholism into genuine epilepsy; this is the case when a person, heretofore only psychopathic and irritable, is seized first in adult life by epileptic attacks, which appear mainly after indulgence in spirituous liquors. Here, arterio-sclerosis of the cerebral vessels often forms the connecting link. From these forms we must distinguish the cases in which acute severe alcoholism and the chronic indulgence in cheap strong spirits (liquors containing fusel oil, absinthe) evoke epileptic convulsions as *acute phenomena of poisoning*. Thus we frequently see, that in delirium tremens epileptic convulsions usher in the symptomatic picture. In such cases, which as *alcoholic epilepsy* must be distinguished from real epilepsy, no more epileptic symptoms appear after cessation of the alcoholism; abstinence is a perfectly sure cure for alcoholic epilepsy, just as getting rid of the morphine habit is for the spasms of morphine poisoning.

While therefore the real cause of epilepsy is an inborn predisposition of the brain, the nature of which is as yet unknown, in the course of the patient's life many *secondary-determining causes* are active, upon which the appearance of the individual attack depends. Injuries of the head or other parts of the body, fright or other severe psychic disturbances, physical and mental overexertion, febrile diseases, masturbation, intestinal worms, foreign bodies in the nose and ear are more or less justifiably regarded as such exciting causes. Occasionally the attacks appear during coitus. Frequently menstruation evokes an attack; this may occur with such regularity, that one may speak of a "menstrual type of epilepsy." Sometimes the first epileptic symptoms appear during or following acute infectious diseases (scarlet fever, typhoid); but in these cases there is always the suspicion, that it may be a matter of symptomatic epilepsy due to infectious encephalitis. Recently, since some observers reported having found the cause of the individual attack in *anomalies of metabolism*, the causal importance of disturbances in digestion has been emphasized. Numerous observations and some chemical investigations, as a matter of fact, seem to prove that an acute poisoning of the brain with some toxic, disintegrating matter, is at the basis of the attack. Of what nature these substances are has not yet been definitely proven (ammonium carbomate, uric acid, cholin?). Of course all these toxic theories fail to explain precisely the most important facts in the study of epilepsy (periodicity of attacks, polymorphism of symptoms, fundamental importance of heredity, symptomatic similarity with non-genuine epilepsy following encephalitis, cranial traumatism, etc.). It is difficult, moreover, to understand why in the individual patient the epileptic attack occurs always under the same conditions. Thus, as has been stated, many epileptics have attacks only during sleep; in others, psychic factors (fear, anger) or mechanical stimuli evoke the attack.

The significance of syphilis, arterio-sclerosis, of some cardiac diseases for epilepsy has not as yet been fully explained. No doubt in so-called *epilepsia tarda,* it is frequently a matter of chronic cerebral diseases, upon an arterio-sclerotic or a cerebro-syphilitic basis, in which epileptic attacks are only a symptom, and which must be distinguished from genuine epilepsy, but in other cases, in symptoms and course, the picture of genuine epilepsy predominates throughout, so that in the present condition of our pathologic-anatomical knowledge it is not possible, to distinguish clinically such manifestations of late epilepsy as separate forms.

The relations of *rachitis* to epilepsy are not yet clear, but it has been reported from various sources, that many epileptics were formerly rachitic, and not seldom they have rachitically-formed craniums. Epilepsy has also been brought into connection with diseases of the thyroid gland; as a matter of fact, we occasionally see epileptic attacks appear in cachexia strumipriva, and in cretinism, which then are to be regarded as symptoms of severe metabolic disturbances.

The convulsions in kidney disease (uræmia), in liver trouble, and in diabetes have nothing to do with genuine epilepsy.

The *first signs of the disease* appear as a rule in the second decade of life; but the attacks and depressions (the latter are occasionally present before the appearance of the developed attacks) may become manifest even in the first decade. If the first signs of the disease do not appear until after the 20th year, acquired injuries, especially traumatisms and misuse of alcohol usually play the excitant rôle.

"*Reflex epilepsy*" constitutes an obscure chapter in the study of epilepsy. After injuries to the limbs, cranium, face, which leave scars, there occasionally arises an epilepsy, in which the motor or sensory aura, that precedes the convulsive attack, emanates from the injured part of the body; the patient perceives a pain, a queer feeling in the vicinity of the scar, there appear twitchings in the limb, and then the convulsions, which are mostly only partial, set in; their outbreak may occasionally be prevented, if the patient quickly ties something around his limb. In such cases attacks may also be evoked by pressure on the scar (epileptogenous zone). Sometimes there appears in the scar upon the skin a neuroma or a small nerve branch. Consciousness is lost only at the height of the convulsions. It is questionable whether this form of partial epilepsy is actually identical with genuine epilepsy. Some of the cases described under the name reflex epilepsy, probably belong to hysteria; in other cases symptomatic epilepsy was present due to local cerebral disease, and only in part of the cases does the assumption seem justified, that the cases are really genuine epilepsy upon an hereditary degenerative basis in which only the excitation of the disease and of the separate attack, as well as the onset of the local symptoms of attack are influenced by traumatism. Always, when the attack in its *entire course* bears the character of *cortical epilepsy*, the supposition lies near that the convulsion is the symptom of a circumscribed disease of the cortex or the cerebral membranes, but not the expression of genuine epilepsy.

Pathological anatomy.—The designation of epilepsy as a neurosis, i. e., as a functional nervous disease, in which no anatomic changes are demonstrable, is to-day no longer permissible. But we must confess, that a pathologic anatomical change, which would be characteristic of all cases of epilepsy, is still lacking. It seems, that even today we combine various pathological processes under the name of genuine epilepsy, and it is not improbable, that the future will bring us a differentiation of epilepsies on an anatomic basis. If one considers all the anatomic changes of the body, observed and described in epilepsy, it is evident without further discussion that taken individually they are of very different importance. Some are purely accidental and insignificant; others are the results of severe attacks (for instance hemorrhages of the skin and the mucous membranes, scars after injuries, possibly also acute vascular changes and small hemorrhages into the brain), others again are signs of general degeneration, which occur in similar manner in other degenerative processes. And finally there are found in the central nervous system chronic changes, in which one may probably see the anatomic foundation for the permanent symptoms of epilepsy (degeneration of character, dementia). On the other hand, even at this time we do not yet know what occurs *in the brain itself* during the *epileptic attack*. One may probably be justified in the supposition, that an excitant process—probably on a toxic base—in the cortex of the cerebrum (change in consciousness, clonic convulsions) and in deeper parts (stage of tonic convulsions) brings forth the convulsive symptoms of epilepsy. The fact, that very different general and local diseases of the brain may evoke epileptic attacks, proves that in the attack a—to a certain extent preformed— "discharging" mechanism is concerned, the excitation of which may be effected by various cerebral stimuli. Complicated motor disturbances in epilepsy (walking and running motions) are brought into connection with an irritation of subcortical centers (thalamus opticus, quadrigeminal bodies?). The nature of the so-called "epileptic change" is not yet known.

From the pathologic-anatomical standpoint, *Alzheimer* came to the conclusion, that what we to-day call epilepsy, is a group of various diseases, a conclusion which, as is well known, *Féré* also reached from purely clinical considerations. Alzheimer differentiates six groups of different processes. The first group comprises *genuine* epilepsy, the causes of which we do not as yet know. In the second group, he assumes external *poisons* as causes, (alcohol, lead) and finds changes in the brain, that correspond to the effects of these poisons. The third group is *syphilitic* epilepsy, dependent on endarteritis syphilitica of the smallest cortical vessels. The fourth form is *arteriosclerotic* late epilepsy, in which one can discover the typical findings of arterio-sclerosis of the brain. The fifth form is symptomatic epilepsy in *focal diseases*, especially in encephalitis. The last group, finally, is epilepsy in case of inhibition of the development of the brain (stadium verrucosum of the brain, tuberous sclerosis). Since by epilepsy, we wish to designate only genuine epilepsy, we shall describe here only the anatomic findings of the first group. In 50–60% of these cases, there is found sclerosis of Ammon's horns, in which, however, Alzheimer sees only a secondary phenomenon of epileptic degeneration, not the real foundation of epilepsy. Gliosis of the margins of the cerebral hemispheres seems to him more important. Medullated fibres and ganglion cells decrease in number; many ganglion cells appear abnormally small; the vessels show slight proliferation, their walls are sometimes considerably thickened. In the lymph sheaths mast cells are often found. If death occurs in the status epilepticus, one finds severe acute changes in ganglion cells and axis cylinders, many of the glia cells show nuclear division, sometimes large amœboid glia cells, which are loaded with the products of decay are seen, furthermore, recent changes in the blood vessels, extravasation of blood, hyperæmia, and edema. The marginal gliosis, should probably be looked upon as the anatomic foundation of the dementia, the acute changes of the parenchyma as those of the attacks. Less frequently than this marginal gliosis, there is found in genuine epilepsy an atrophic sclerosis of entire convolutions or of an entire hemisphere. In such sclerotic parts of the brain the nervous tissue is seen to be very considerably wasted; the third cortical layer in particular is very atrophic; the glia proliferation extends to the marginal layer and the medullary lamina. The marrow is seen to be filled in fresher places with little fatty granular cells, which spring from glia cells. In the status epilepticus, Alzheimer sees the clinical expression of the height of acute destructive cortical affection. He assumes that this cortical disease *could advance even during the time, that is free from attacks.* This would explain the unquestionable fact, that epileptic dementia is by no means always proportioned to the number and severity of the attacks.

Of the numerous other anatomic findings, that have been discovered in epileptics, the following need mention: sometimes the cranial bones are thickened, there are exostoses in the cranial vault, the cerebral membranes are adherent. Frequently the cranium exhibits signs of former rachitis. We have already pointed out the numerous signs of degeneration, that are found in epileptics. Occasionally an abnormally small aorta is supposed to have been found. Attempts have been made to make a pathological narrowness of the vessels of the brain responsible for the origin of epilepsy. But all certain proofs are still lacking for this. In very many epileptics, one finds even in middle age pronounced arterio-sclerosis, regarding which, however, we must consider, that many epileptics are chronic drinkers. Parenchymatous fatty degeneration of the muscles of the heart, liver and kidney were found in cases, in which death occurred in the midst of acute epileptic phenomena. Disease of the sympathetic in epilepsy has been repeatedly reported, but not yet anatomically demonstrated. Likewise, it must still be considered questionable, whether a true hypertrophy of the brain (weight up to 2800g!), which is supposed to destroy the normal relation between the brain and the cranium (*Anton*) may be considered as a basis for some cases of epilepsy. Circumscribed hemorrhages of the skin, or internal organs (pleura) are referred to central vaso-motor processes; more frequently they are phenomena of congestion.

The **diagnosis** of epilepsy is confronted by various problems. Is the symptomatologically indubitable "epileptic attack" a symptom of the disease "epilepsy," or of another cerebral disease, or toxæmia? Are the attacks described or observed of an epileptic or hysteric character? Is the dizziness an attack of petit mal, or a symptom of neurasthenia or of a disease of the heart? How are the depressions and transitory psychoses of the epileptic differentiated from other emotional disturbances? What may the chronic mental state of epileptics be confused with? How is it differentiated from the degeneration of hysteric and other psychopathic patients, how from congenital weak-mindedness, or from non-epileptic dementia? Some of these questions have already been discussed in the symptomatology and the ætiology of the disease, others shall be again opened here; with regard to psychiatric differential diagnosis, the psychiatric textbooks must be consulted.

First, we must always remember the fact that, as has already been stated, typical epileptic attacks occur in many nervous and mental diseases; the exact neurological and ophthalmoscopic examination of the patient will usually disclose, whether it is a question of genuine or symptomatic epilepsy. The diagnosis of incipient tumor of the brain may cause especial difficulties. The epilepsy, that appears after encephalitis, may easily be mistaken for the genuine form, if the acute process has left behind no uncertain focal symptoms (infantile paralysis). The same is true of many cases of traumatic causation. Since the treatment of the attacks, that appear after encephalitis, is not different from that in genuine epilepsy, a mistake in diagnosis is here of little importance. It has been recently pointed out repeatedly, that in psychopathic persons, isolated epileptoid attacks occur (*Oppenheim's* "psychasthenic convulsions," *Bratz's* "emotional epilepsy"). In these cases the rest of the clinical picture, especially the analysis of the entire personality must be considered for the correct diagnosis. *Heilbronner's* "accumulated small attacks" are also probably essentially different from true epilepsy. Eclampsia of infants in the first year of life, which is to be traced back to metabolic disturbances, has been entirely separated from genuine epilepsy by the exact investigations of *Thiemich* (pathological increase of the mechanical and electric, especially of the galvanic excitability of the peripheral motor nerves), although later examination of formerly eclamptic children proved, that many later suffer from nervous diseases, some (*Heubner*) become epileptic. Occasionally, in the convulsions of children, acute serous meningitis, which may be established by lumbar puncture, is present; more frequently there are relations with tetany.

In *late epilepsy* (appearance of attacks only after the 40th year of life) one must look for the signs of arterio-sclerosis, as well as ætiologically for syphilis, alcoholism, lead poisoning. One must emphasize, that this late epilepsy too may appear under very manifold clinical pictures. Petit mal, twilight conditions occur in it also.

Migraine may be confused with epileptic equivalents, and authorities are not lacking, to point out the close relation between the two diseases. It must be emphasized, however, that migraine shows no tendency to steady progression, that it nearly always leaves the psychic personality intact, that (in contradistinction to epilepsy) it is more common in women than in men, and that in it the inheritance is according to sex (*Möbius*). Common to both, on the other hand, is the periodicity of attacks arising from an endogenous cause.

We have already pointed out the difference between epileptic convulsions in acute alcoholic poisoning, and epilepsy excited and aggravated by the misuse of alcohol.

The simple "*faint*" is distinguished from the epileptic loss of consciousness, in that it frequently has a direct physical and psychic cause, (heart disease, terror, heavy loss of blood), in that the pulse in the usual faint is mostly small and very frequent, occasionally quite irregular; furthermore, the unconsciousness is usually not total (*Oppenheim*). The vertigo of the neurasthenic is, as a rule, connected with psychic excitement, and passes without any disturbance of consciousness worth mentioning, while in epileptic vertigo, consciousness is always distinctly impaired; in the latter, moreover, the position of the body is without influence (*Gowers*).

The differentiation between epileptic and hysteric attacks (*Hoche and others*) belongs to the most important problems of neurology, since a mistake in diagnosis leads to grievous prognostic errors, and therapeutic mistakes. There is no such disease as "hystero-epilepsy;" but epileptics, like all degenerates, may evince hysterical symptoms, and in rare cases, the hysteric attack may symptomatically resemble, or perhaps be identical with the epileptic attack. In spite of this, they are essentially different from all other standpoints. For clinical differentiation, one should remember: the typical epileptic attack usually arises without any psychologically demonstrable cause, its movements are severe "organic" convulsions, or impulsive automatisms, in which no psychic processes find expression. It is often characterized by an obvious aura, the pallor at the beginning, the subsequent cyanosis, the loud inspiratory cry, the usually headlong fall to the ground in total unconsciousness, the independence of the course of the convulsions from external influences, the succession of tonic and clonic spasms, the bitten tongue, the appearance of bloody foam at the lips, the loss of pupillary reaction, the short duration of a few minutes, the heavy waking with headache, a morbid desire for sleep, general muscular fatigue, amnesia for the attack itself. The hysteric attack is quite different. Often it can be shown, that it is the reaction to a psychic excitement, that psychic processes are responsible for its excitation and further development. Sometimes it may be evoked by suggestion. The disturbance in consciousness is not so severe, does not appear so suddenly, so that the patient as a rule, finds time to sit down first, or throw himself safely on the ground, whereby injuries are nearly always avoided. There is something voluntary in the movements of the attack (cf. the chapter on hysteria); they are, as a rule, movements of expression; the patient even speaks or screams during the attack itself. The attack is usually susceptible to external influences, so that the physician may place himself in connection with the patient during the attack, and influence by suggestion the course of the individual phases. The pupillary reaction is usually not lost, though spasmodic conditions in the sphincter occur, possibly also in the dilator pupillæ during the hysteric attack (miosis or mydriasis); in such cases, not only the reaction to light but also that of convergence is lost. But in the majority of cases, the old diagnostic principle holds good, that the pupils in the epileptic attack do not react, but do react in the hysteric attack.

For the patient to bite the tongue is rare during the hysteric attack; more common are complicated movements of biting, which are evidently psychically conditioned. The self-inflicted injuries in the attack of the epileptic are unfortunate accidents, those of the hysteric usually voluntary, and therefore more complicated actions. Involuntary discharges during the attack are rare in hysteria. Hysteric attacks are of much longer duration, they often last for hours; when the patient wakes from them, he is surprisingly well. There is rarely total amnesia. In epilepsy, the effects of the attacks on the general health are serious, if the attacks occur frequently, whereas hysterics may suffer daily with long attacks, without any injury to the general state of their health.

Finally—and this is of especially great importance when the physician himself has not seen an attack—the permanent mental condition of the epileptic differs totally from that of the hysteric or psychopathic. Hysteria and mere psychopathic conditions never lead to mental dullness. The hysteric character has of course some symptoms of degeneration in common with the epileptic (moral depravity is common to both), but is distinguished from the other in essential traits (mental activity, intelligence, memory, etc.). The character of hysteria is in general that of increased suggestibility; that of epilepsy a narrow or weak-minded inflexibility. Where it can be shown, therefore, that the patient in the course of a disease accompanied by convulsions has experienced a change in personality, corresponding to that described above (p. 723), the presence of epilepsy should be assumed even though the kind of attacks remind one more of hysteria; and where, on the other hand, a convulsive disease, existing for years, and accompanied by very frequent attacks, has not noticeably injured the psychic personality, the probability speaks far more for hysteria, even though the attacks appear symptomatically to be epileptic. The conjecture has been entertained (*Jolly*, *Karplus*, *Hoche*), that hysteria is able to produce the cerebral condition, the nature of which is as yet unknown, which must be supposed to exist in the presence of epileptic attacks. The epileptic attack in its genuine form (unconsciousness and muscular twitchings) presupposes the performance of certain processes in the brain which always return in the same form; the cause of the individual attack need not lie in any coarse material changes in the brain, so that the possibility that in degenerative, or hysteric conditions, the increased excitability of the brain may, under certain conditions reach the stage, in which the "epileptic" convulsive discharge usually takes place, can not be excluded. It will be possible then, that, in such exceptional cases, the attack of the hysteric is symptomatically identical with the classical attack of the disease epilepsy (*Hoche*). The same observation may hold good for *Oppenheim's* psychasthenic attacks also.

The *simulation of epilepsy* presupposes knowledge of the course of an epileptic attack. In the simulated attack, the pupillary rigidity is lacking, as well as the transitory loss of the tendon reflexes, likewise the originally pallid discoloration of the face, which later in the convulsive period, passes over into deep cyanosis and the increased secretion of nitrogen in the urine. Biting of the tongue, dream-like confusion, or deep sleep after the attack militate against simulation. The epileptic is totally analgesic during the attack, the course of the convulsions is independent of external influences, while the simulator as a rule, shows neither analgesia, nor is he totally uninfluenced by external factors. If the physician has not seen an attack, he must be very careful in stating that the description proves the presence of epilepsy; in such cases, it is particularly important to look for the permanent symptoms of epileptic degeneration. The mental dullness and prosiness of the epileptic contrast with the deliberate and conscious way, in which the simulator describes his "attack." One is always astonished at seeing how little epileptics, who suffer from attacks for years, can say about their course, even when they could have obtained information on the subject by asking the people about them.

Prognosis.—The prognosis of the disease must always be made with the greatest caution. One is usually far too ready to assume the possibility of a cure. The earlier the attacks begin, and the earlier the mental faculties are impaired, the worse is the prognosis. On the other hand, it is wrong to conclude from the *severity* of the convulsions, that the course will be unfavorable. Experience teaches, that the patients with petit mal, with incomplete attacks and twilight conditions become imbecilic, just as well as patients, who suffer from typical severe attacks. It even seems, that the result of treatment, especially of treatment with bromides, is less favorable in cases with long intermissions, than in the form with frequent fits. Complete permanent recoveries occur, but are rare (probably not 10%). Even after intermissions of several years, attacks may recur. The prognosis of the disease depends also largely, upon whether the patient avoids or does not avoid intoxicating liquors. Epileptics, who drink much, may not hope for a cure of their disease. Grave hereditary predisposition, very early appearance of the first symptoms, complications with idiocy or moral dementia, make the prognosis less favorable. If the attacks occur in mentally normal patients, only once a year, or even less frequently, the *permanent* symptoms, that make epilepsy so grievous a disease, may remain entirely absent.

As a rule, the attacks appear approximately periodically (about every 8 to 14 days; during the menses); sometimes however there are free intermissions of months and years. The attacks of petit mal usually recur far more frequently (in many cases, several times a day) than the severe convulsions. Epileptic women occasionally remain free from attacks during pregnancy. It has been known also for a long time, that attacks are absent during grave acute diseases; it has even been believed, that a cure for epilepsy might be attained by the influence of febrile diseases—an influence, the nature of which is absolutely obscure—just as was asserted about progressive paralysis. About half of all epileptics die in the status, or perish of injuries sustained in the attack; some suffocate during the attack. When there is a high degree of idiocy and marked uncleanliness, infections and decubitus may endanger life. The mean duration of life is far shorter than in healthy persons. Only about three per cent. reach their fiftieth year. Many epileptics die of tuberculosis.

Treatment.—There is no single agent, which may be considered a cure for epilepsy. The causes of the disease are too little known to permit of a causal therapy. In epilepsy, as in all other diseases, the most important task is the prevention of its development. Everything, that favors degeneration, favors the development of epilepsy. Drunkenness of the parents is especially ominous. Therefore he, who wishes to beget children, should refrain from the misuse of spirituous liquors. Mentally abnormal, epileptic, hysterical persons should not marry and have children; but if they do marry, they should see to it at least, that the other party to the contract is nervously sound. Converging predisposition frequently creates severe epilepsy. The nourishment and bringing up of the small child should be governed by the rules of hygiene. Epileptic mothers should not nurse their children themselves. It is a crime, to give a child intoxicating liquors. If symptoms of hereditary predisposition appear in the physical or mental realm, special care is necessary. Pathological parents should not be permitted to look after the education of their children. Over-feeding the child with meat increases the excitability of the nervous system and favors the origin of convulsions. Even though eclampsia and epilepsy are not essentially the same, experience teaches, that many former eclamptic children later become epileptic. Therefore, one should avoid nutritional disturbances at an early age. If traces of rachitis appear in a child, one should treat it immediately according to the principles laid down by the authorities in children's diseases. Hardening the somewhat older child, avoidance of mental shocks, avoidance of masturbation are important tasks in the education of threatened children.

If the disease has broken out, it is a question of establishing with certainty, what factors have cooperated in its origin. One should investigate the hereditary conditions carefully, permit the prodromal symptoms, the aura, the course of the disease to be described exactly, seek out past diseases, and attempt to discover clearly, whether an organic cerebral disease, a metabolic disease, or an injury, may be concerned as a cause. In investigation of the anamnesis, one should refrain from suggestive questions, and not forget, that most laymen are poor observers of disease phenomena. If one is sure, that epilepsy is present, it is necessary to judge the severity of the disease (frequency of attacks, condition of the mental faculties), because the treatment should be directed according to the severity of the disease.

Treatment of the attack.—The patient must be so laid, that he can not injure himself in the convulsive stage; during the clonic spasms, one must not try to hold his limbs; restricting clothing (collar, corsets, belt, etc.) should be unfastened; if possible, a piece of cork, wood, or rubber, should be inserted between the teeth, to avoid severe bites of the tongue and the lips. Saliva and mucus should be removed from the mouth. After cessation of the convulsions, one should allow the patient to lie quietly or put him carefully to bed, but *not* wake him from sleep. The patient should remain in bed for the rest of that day, and after severe attacks, for the next few days, and have physical and mental rest. The people about him should avoid showing him their sorrow and fear. Children ought not to see epileptic convulsions.

Treatment of the disease itself.—The principles of the treatment of epilepsy must be: preservation of the brain from all injuries, in fact from everything that might increase its excitability; avoidance of all nerve poisons; regulation of the mental and physical activity, according to the strength of the patient; administration of medicinal measures, which according to experience lessen the excitability of the brain. In detail, we may say: since, on the whole, the frequency and the severity of the attacks afford a means of measuring the severity of the disease, and since, above all, the mental deterioration appears preferably, if not exclusively, in patients, who have many attacks, physicians have long perceived the main task of treatment to be the attempt to make the attacks disappear, or at least, to diminish their number, though it can not be denied, that occasionally the absence of attacks is felt subjectively in a distressing manner by the patient, so that he even longs for an attack (*Binswanger*). Since some epileptics have attacks preferably after certain causes (psychic excitement, physical overexertion, coitus, overloading the stomach, etc.), the point is to avoid these causes as much as possible. The patient is constrained to live according to the rules of mental hygiene; he needs plenty of sleep, also work in proportion to his strength. Should an epileptic child have frequent attacks during the day, it must be taken from school; so long as his mental faculties are not clearly impaired, he may have private instruction; but as soon as a considerable deterioration in intellect appears, mental development must make way for physical care and treatment in an institution. One must be careful in the choice of a vocation; work, in which a sudden attack endangers life (bricklayer, roofer, smith, etc.) is to be avoided. The same holds good for vocations, that lead to the misuse of alcohol (hotel-keeper, brewer, waiter), and of those that demand work in the hot sun. Since an epileptic attack is apt to terrify other people very much, epileptics are not willingly retained in service; therefore it is advisable to choose a calling, in which the patient may work by himself as much as possible, and is withdrawn from association with others. This is advisable also because the epileptic is frequently irritable, in his dealings with other people exacting and sensitive.

This rule holds good from of old, that the *diet* of the patient should be non-irritating, predominantly consisting of vegetables. According to the wide experience of *Alt* and others, milk and vegetables are better digested by most epileptics than meat. In cases with night attacks but little should be eaten in the evening and that easily digestible. The use of intoxicating liquors is strictly forbidden every epileptic. Also coffee, tea and smoking are believed to be injurious, as well as strong spices, bouillon. Since it is possible, that putrefactive processes in the intestines excite epileptic attacks, constipation and intestinal catarrh should be overcome (calomel, rectal injections of oil, etc.).

Should the attacks be very rare (about one a year), the *use of medicines* may for the time be dispensed with. But usually it will be advisable to employ medicines. Among the large number of drugs, that have been recommended in the course of time, most have proved failures, so that they have only an historical interest. We shall not attempt to name them here. Most successful, in the experience of most specialists, are the *salts of bromine*, which may be administered for years without causing any injury. I prefer sodium bromide in 10% solution, given in carbonated water, to all other remedies. Children should be given 30–60 gr. pro die, adults, 60–100 gr.

according to the frequency and severity of the attacks. Epileptic psychoses may make doses of 60–120 gr. per day necessary. Bromide acne should be combated by the internal administration of arsenic (*Fowler's* solution, 5 to 10 drops twice daily). Treatment with the bromides should never be suddenly interrupted, since in that case accumulated attacks appear quite frequently. Bromides must be given for years. Erlenmeyer's mixture (Kalium bromatum 10.0, natrium bromatum, 10.0, ammonium bromatum, 5.0), and Sandow's effervescent bromides, are also favorite medicinal preparations; they, however, have no special advantages. Recently bromipin (bromides and sesam oil) which contains 10% of bromine, and is taken by the spoonful, but is expensive and tastes bad, has been much prescribed. Borax and camphor monobromatum are praised; the latter is supposed to have an especially favorable effect on the attacks of vertigo. To aid the influence of the bromides, it is advisable to put but little salt in the patient's food (Toulouse and Richet, Balint) so that the body lacking sodium *chloride*, may take in the *bromide* the more eagerly. There are certain kinds of bread and zwieback, in which ordinary salt is replaced by salt of bromine.

With the persistent administering of bromides in large doses the danger of bromism is connected, i. e., of chronic bromide poisoning, which may appear in an overfeeding of the body with bromine. Acne, constipation, salivation, emaciation, weakening of the cutaneous and mucous membrane reflexes, loss of the corneal reflex, a feeling of general weakness, difficulty in mental work, *hypochondriac* depression, in high degrees of the poisoning disappearance of memory and difficulty in recollection, are the main symptoms of bromism, which force us to stop the treatment by the bromides. Careful attention to the skin is urged upon every epileptic, who takes bromides.

With the bromide treatment one usually attains in genuine epilepsy a decrease in the number of attacks; only exceptionally, however, their total disappearance. In order to strengthen the effect of the bromide, *Flechsig* first recommended the so-called *opium-bromide treatment*, which sometimes succeeds in permanently getting rid of the attacks. The treatment is not without danger, and should be employed only in strong, not yet idiotic persons, of youthful age under the constant supervision of the physician. Opinions as to its curative value vary. *Kellner* has again urgently recommended it recently on the ground of careful investigations. At first he gives opium in gradually increasing doses for 50 days, beginning with opium 0.05 three times a day, increasing the dose every other day, by 0.01. Thus on the 50th day, 0.29 g is given three times a day. On the morning of the 51st day, he gives 0.03, then he suddenly omits opium entirely, and at noon and in the evening, gives 15 gr. of bromide mixture. In the following days he increases the dose of bromide mixture, 15 gr. per day, so that on the 58th day, he reaches 135 gr. and remains at this dose for some time. During the administering of the opium, he gives a tablespoonful of a 1% solution of hydrochloric acid three times daily, if necessary also bismuth and Carlsbad salt. A light unspiced vegetable diet, remaining in the fresh air, physical and mental rest during the use of opium, and short lukewarm baths (24 to 17 degrees Celsius, duration 10 to 20 minutes) serve as adjuvants to the treatment, the first part of which (opium) should be applied only in a hospital. Deaths were reported by several authors.

In attacks *by night*, it is advisable to give the bromide in the evening in 1 dose (45–60 gr.). The *status epilepticus* demands very large doses of bromide (12 to 18 g); in it the administering of amyl hydrate (90–120 gr.) or chloral hydrate (30–45 gr.) in a rectal injection is a favorite remedy; hyoscinum hydrobromicum (subcutaneously 0.001 to 0.002) may be tried in severe cases, also inhalation of chloroform, bleeding, washing out the intestines.

Besides the preparations of bromide *atropin* (0.0003 to 0.0005) and *extractum belladonæ* are recommended by some. But their effect is very uncertain. It is doubtful, whether cerebrin is of any avail. Digitalis, adonis vernalis, strophantus, have been given in such cases in which epilepsy had to be traced back to disease of the heart, or to arterio-sclerotic and alcoholic cerebral changes. Where syphilis has a causal relation, mercury and iodide of potassium are proper. There is no *operative treatment* of genuine epilepsy. Many suggestions have been made to treat the disease surgically; operators have resected the sympathetic, ligated the carotid, trephined the cranium, removed parts of the cortex. Where any result was attained by such attempts, no genuine epilepsy was concerned, but either symptomatic convulsions in organic diseases of the brain and the cerebral membranes, or hysteric attacks. Where it is probable from the ætiology or the course of the attack, that a circumscribed cerebral disease or an injury to the cranium is responsible for the epileptic symptoms, it is proper, if possible, to remove operatively as soon as possible, the source of irritation; just so in "reflex epilepsy," the scar, the neuroma should be removed. But the uncertainty of the result has somewhat discredited the entire study of reflex epilepsy.

Opinions of physicians vary considerably as to hydrotherapy in epilepsy. Some, like *Ziehen*, recommend a careful cold water treatment, whereas others state, that all rather severe irritations of the skin are injurious. Cold showers over the head are not to be used. No real curative effect can be ascribed to hydrotherapy any more than to electricity. Nor can anything important be expected from hypnotism. If epilepsy leads to serious change in character, to idiocy, if frequent twilight conditions or psychoses of long duration occur, it is necessary, since the patients are a great menace to those surrounding them, that they be treated in an institution. Here, naturally, psychic treatment steps in. But the main value of *treatment in an institution* lies in the impossibility of begetting children. Every epileptic should be advised not to marry, because his progeny are endangered to a high degree.

For youthful patients, who do not yet show mental impairment, special training schools in connection with state or communal sanitariums are recommended. In the latter, the sections devoted to the curable cases should be separated from those devoted to the care of the incurables and the idiots. Some patients with a hypochondriac disposition suffer very much from the consciousness of their disease; rest and encouragement, sympathy and ample advice, are necessary so that the patient may not be driven from the treatment of the physician to that of the quack. It is astonishing what a fine sense even demented epileptics have for the fact whether one has time and sympathy for their physical and mental needs.

Medico-legal importance.—No other nervous or mental disease leads so frequently to criminal actions as epilepsy, and in none is the proof that the perpetrator, in committing the crime, was mentally ill, in the sense of the §51 of the Str. G. B. (German criminal code) so difficult, as it may be under certain circumstances in epilepsy. Some crimes of epileptic patients are the consequence of their permanent general irritability, and their tendency to explosive deeds of violence (insults, destruction of objects, bodily injuries) others are concomitant phenomena of general social deterioration to which many epileptics succumb, as, begging, vagabondage, thefts.[1] Arson, murder, severe physical injuries, immoral offenses of the most brutal kind, among soldiers desertion, are found not only in severe epileptic psychoses, but also in the lighter epileptic depressions, the pathological basis of which is not clear to the laymen without further explanation. The understanding that the crime of an epileptic was committed in the twilight condition, or in the transitory depression, presupposes an exact knowledge of the entire case and broad clinical experience. The physician will do well in all cases in which the possibility of such a connection exists, to advise the examination and observation of the perpetrator in an insane asylum. For the principles governing the medico-legal determination of the crimes of epileptics see the text-books on medical jurisprudence.

The very nature of epileptic degeneration brings it about that many such patients lead a flighty wandering life, becoming *vagabonds*. One always finds epileptics in work-houses.

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[1] Especially numerous and grave crimes are committed by patients with epileptic psychoses.

VI

THE ORGANIC NERVOUS DISEASES OF CHILDHOOD

BY

J. IBRAHIM (Munich)

1. CEREBRAL PARALYSIS OF CHILDREN. (INFANTILE CEREBRAL PARALYSIS)

Hemiplegia and Diplegia Spastica Infantilis

Infantile cerebral paralysis is a *clinical conception.* We designate by this term the final stage of a number of disease processes, different in respect to ætiology and morbid anatomy, which chiefly have one factor in common, namely, that they affect the brain in early infancy or even during fœtal life. Though the clinical picture of infantile cerebral paralysis is very diversified, it is nevertheless impossible to draw, with any degree of certainty, deductions from these symptoms as to the original process of the disease and even the post mortem findings make it impossible except in rare cases to draw a definite conclusion regarding the initial process, which frequently precedes the fully developed phenomena by many years. To classify in separate groups this wealth of disease pictures from the point of view of ætiology or morbid anatomy is therefore an impossible task; but, on the other hand, a strict division according to clinical types is also out of the question, as an almost gapless series of transitional forms connects one type with the other. Nevertheless an attempt to pick out of this chaos of clinical cases special clinical groups must be of considerable practical value, the more so as, to a certain extent, these separate groups, even from the standpoint of ætiology and prognosis, show some features, though possibly not very far-reaching ones, in common.

Etiology.—We know quite a number of factors, that play a certain *predisposing rôle* in infantile cerebral paralysis. The most important are psychoses and nervous diseases, phthisis or syphilis in the ancestors or alcoholism in the father. With comparative frequency firstborn children are stricken (*Gowers*) or children that in a long series of brothers and sisters are born last or late (*Freud*). Besides these predisposing factors, which not infrequently are the only ætiological factors which we can elicit in the individual case, there are also noxæ, which may be regarded in many cases as the *direct causes* of the disease, especially two, traumatism and infectious disease. *The trauma* may affect the gravid uterus, it may, in the form of cranial traumatism lead in early or later infancy to a hemorrhage in the brain and to cerebral paralysis, but it plays its most important rôle in the ætiology of this disease in the act of parturition itself. Difficult or protracted deliveries, doubtless, are in many cases the cause of the cerebral paralysis making its appearance in the child, a shorter or longer period after its birth. In these cases meningeal hemorrhages appear, which injure the motor cerebral centers and, according to clinical experience, usually cause bilateral lesions. That the use of the forceps may be followed by similar consequences will hardly be denied, but it is certain, that instrumental aid far more frequently prevents the hemorrhage, than causes it and that the circumstances, that necessitate the use of the forceps, are the main cause of the origin of the disease. Of the noxæ occurring during child-birth *asphyxia* certainly deserves special consideration.

Psychic trauma, fright during gravidity, seems also to need to be considered ætiologically in some cases.

Many authors attribute great importance to *premature birth* and it is a well known fact, that a large number of children, especially with paraplegic rigidity, but also those afflicted with other diplegias, are children, that have been prematurely born. The difficulty of the question is, however, whether in these cases premature birth is always to be regarded as the cause and not much more frequently as the consequence of the disease, whether the same causes, which underlie premature birth, are not also the causes of the cerebral paralysis (for instance syphilis). We certainly know, that in many cases it can be proved afterwards, that such prematurely born children show cerebral changes, the origin of which is surely to be dated back to a time long before birth. The same knowledge however can be gleaned also in cases of difficult labor. We are not able to state how often in such cases mistakes are possible.

Another important source of the origin of infantile cerebral paralysis are *infectious diseases*. That syphilis in the congenital cases, as well as in the postnatal ones, may play a very important rôle can no longer be doubted in consideration of the more recent reports. *Fournier* regards the paraplegic rigidity as a parasyphilitic affection. Affections after the first year, however, show not rarely an ætiological connection with various acute infectious diseases (measles, scarlatina, typhoid, pneumonia, variola and others). It is likely that, as a rule, vascular brain lesions (embolisms and hemorrhages) follow these diseases.

Whether there is a separate form of infantile hemiplegia, which could be classified as an infectious disease sui generis, is a much mooted question. *Strümpell* in 1884 upon clinical grounds expressed this opinion in a rather convincing way and tried to separate these cases as *polioencephalitis acuta* or *encephalitis acuta* from the general form of infantile cerebral paralyses. He referred in this respect to the analogy with acute poliomyelitis (as *Vizioli* had done before) and pointed also to the fact, that occasionally of two children of the same parents, one fell victim to spinal, the other to cerebral paralysis. Besides, combinations of flaccid and spastic paralyses in one and the same patient have been described. Such cases, however, are extremely rare; but, on the other hand, exact observations made during the last great epidemics of poliomyelitis acuta in northern countries have proved, that the cerebrum with relative frequency takes part in the inflammatory process (*Wickmann*, *Harbitz and Scheel*), so that hardly any doubt can exist about the plausibility of *Strümpell's* theory, which was indorsed by *Pierre Marie* and many other authors. Still it must be regarded as remarkable, how extraordinarily rare in those large epidemics hemiplegias of the character of infantile cerebral paralysis are found and therefore it must rather be left undecided, whether a really important rôle for the origin of hemiplegias can be assigned to encephalitis. As yet an anatomic knowledge of such an early stage of our disease has not been gained. It is true that recently several cases of encephalitis in infants have been investigated during the height of the inflammatory process (*Fischl*, *Ganghofner*, *Weyl*) but the clinical picture in these cases did not show at all that a typical infantile cerebral paralysis did develop out of the grave disease that caused the death of the infants. Therefore, there is still left a wide field of research in this regard.

Finally it is to be mentioned that *convulsions*, eclamptic attacks and the congestions resulting therefrom are regarded by some investigators as direct determining causes of cerebral paralyses (*Sachs*, *Osler*) while others, especially *Freud*, regard the spasms always as an expression of an already existing cerebral affection.

In the individual cases it may be frequently quite impossible to trace the disease to a definite cause; very frequently also we find a combination of several predisposing factors in one and the same patient; the important factors of difficult delivery (protracted parturition), asphyxia and premature birth are designated after their discoverer as *Little's ætiology;* they are mostly found in the anamnesis of the diplegic types of paralysis.

Finally it must be mentioned here, that following *Sachs* the c rebral paralyses of infants are sometimes divided from the viewpoint of ætiology into three large groups, *cases originating before, at, and after child-birth.* Doubtlessly it is of great interest to find out in any case, how and when the disease originated. But very frequently all endeavors will be fruitless, especially in the cases, in which the paralysis is observed in the course of the first year, as these could be justly classified among any of these three groups, unless an acute stage clearly testified to the onset of the disease.

Pathological anatomy.—The anatomical findings of infantile cerebral paralysis are very diversified. What we know so far are only the processes, after they have run their course, the terminal stage of the processes, while our knowledge of the *initial stage of these lesions* is very scanty. Of these in fact we are better acquainted only with the hemorrhages caused by injuries during child-birth; we know, that they are occasioned, as a rule, by the tearing off of the veins before they enter the sinus (*Virchow*). But here, too, we know only the anatomical picture of the cases, that ended lethally, without being able to state with certainty, whether in case of recovery a typical cerebral paralysis would have been developed. The transformation of a clinically definite meningeal hemorrhage into a diplegia has not been observed as yet, so far as my knowledge goes, but a few post-mortem findings are known (*MacNutt*, *Railton*) which can be explained with reasonable certainty as consequences of hemorrhages of the surface. There is besides good reason for the assumption, that the majority of cerebral paralyses in infants are conditioned upon *vascular lesions* (*hemorrhage, embolism, thrombosis*), that part are caused by *inflammatory processes* and possibly a small part through *primary degenerative processes.*

If we take into consideration the *morbid anatomy of the cases that have run their course*, we find in part localized injuries, as *softening foci*, *cicatrices*, *cysts*, but with special frequency *defects of the cerebral cortex*, which cause more or less deep, frequently funnel-like depressions. Since *Heschl* these defects are designated as *porencephalias* (Fig. 189). They may communicate directly with the ventricles, may be unilateral or bilateral and show a predilection for localization in the region of the motor centers. Without doubt the majority of the porencephalias have a prenatal origin and are to be regarded as anomalies in development (*Kundrat*), but an extra-uterine, for instance traumatic (*v. Kahlden*) or embolic origin (*Heubner*) is certainly possible. In spite of the many criteria, which have been brought forth, it is not possible to state with certainty the origin in each individual case. The same must be said of another finding, which may combine with porencephalia, viz. *microgyria* (see Fig. 189). By this term is designated a fine wrinkling of the cerebral cortex, which may be explained as an anomaly in development or as a phenomenon of contraction following meningeal hemorrhages or meningo-encephalitis (*Oppenheim*).

The cerebral membranes frequently show cloudings, indurations, adhesions, in the region of the cerebral affection. That cases of true meningitis, from which the patients have recovered, may also show the clinical picture of infantile cerebral paralysis is certain, but such cases ought not to be classified with our disease, no more than those of pure hydrocephalus.

Besides the focal affection there is found usually a more or less clearly defined wasting of the entire cerebral hemisphere (*atrophia cerebri*, cf. Fig.

189); it is the result of a sclerotic process, which may develop from the most diversified initial disturbances (hemorrhage, thrombosis, encephalitis) and shows with remarkable frequency a decidedly progressive tendency.

Not infrequently no localized affections at all can be found, but large parts of the brain show signs of induration (*lobar sclerosis*) or there exist bulb-like indurations (*tuberous sclerosis*). Furthermore the brain may appear entirely normal macroscopically and only the histological investigation is able to find alterations of nerve cells and proliferations of the glia.

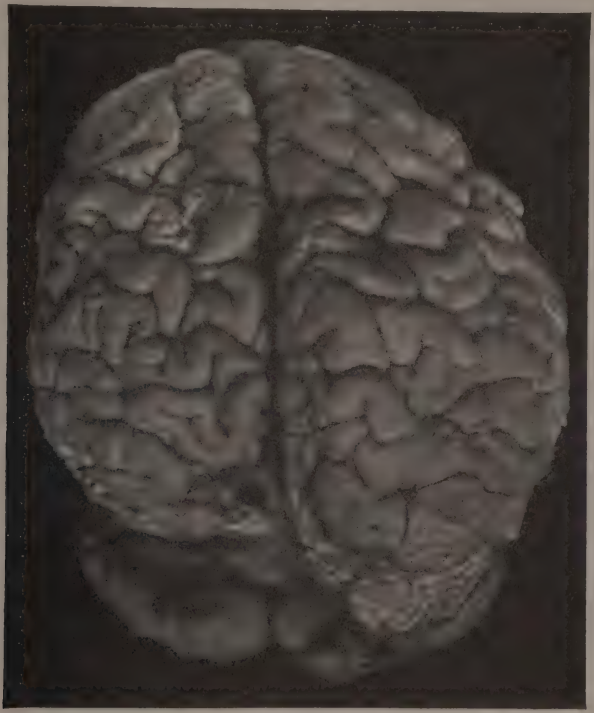

FIG. 189.—Brain of a child, 3 1/2 years old with congenital infantile cerebral paralysis. Porencephalon, specially in the region of the left occipital lobe. Microgyria, principally in the left frontal lobe. Atrophy and sclerosis of the left cerebral hemisphere. (From a preparation of the pathologic institution in Munich.)

It seems, that simple inhibition in the development of the cerebral cortex, too (*agenesis corticalis, Sachs*) or *primary degenerative processes* may produce clinical pictures, which have to be reckoned with those of infantile cerebral paralysis (*Mya and Levi, Collier*).

In the prenatal cases other serious developmental inhibitions of all sorts may be present in the brain, which can not be spoken of here to any extent. Of special interest with regard to the pathological physiology of muscular rigidity is the condition of the *pyramidal tract*. As a rule it proves to be atrophic, degenerated, or at least impaired in development and the medullated sheaths of the nerve fibres imperfect. But it must be mentioned that cases of general or paraplegic rigidity are known, in which the pyramidal pathways show no alterations whatsoever.

Symptomatology.—A number of characteristic clinical symptoms betoken infantile cerebral paralysis. They may appear in a more or less pronounced degree in all forms of the disease, and there is not one of them, which could be ascribed exclusively to one definite group. The principal symptoms, which must be mentioned here are, besides the paralysis, which may play a very insignificant rôle in the clinical picture, the rigidity and the contractures in the paralyzed limbs, the concomitant associated movements and spontaneous movements (chorea and athetosis), the disturbances in growth, weakness in intellect and epilepsy. Notwithstanding this a separate presentation of hemiplegia and diplegia seems appropriate, as the two clinical types, in spite of frequent transitional forms, in clearly defined cases materially differ one from the other.

Hemiplegia Spastica Infantilis (Unilateral Cerebral Paralysis of Children)

The hemiplegic cases are generally distinguished from the diplegic ones, in that the majority of the former are of postnatal origin, that the arms are usually more seriously affected than the legs, and that they very frequently lead to disturbances in growth and to epilepsy.

In typical cases, the course of the disease is about as follows: Children in the earlier years of childhood, sometimes following an infectious disease, at other times in the full bloom of health, become sick with fever, sopor, vomiting and convulsions; after the acute stage, which is usually of short duration, has passed, a more or less complete hemiplegia, in which the face, as a rule, participates, is usually noticed. The paralysis is at first flaccid, but soon becomes spastic; aphasia, which is often present at the beginning, disappears. The paralysis retrogresses later on considerably, especially in the legs, while the arm frequently is injured more severely and shows marked contractures. With the retrogression of the paralysis, frequently athetoid and choreic phenomena appear in the paralyzed half of the body. Later this side shows a restricted development in length as well as in thickness. In many cases, after weeks, months and even years, epileptic attacks appear, which, as a rule, increase in frequency and severity. Even in those children who are free from convulsive attacks, the mental faculties are more or less seriously impaired.

If we enter into the details of the disease picture, we find in regard to the **ætiological factors,** that congenital cases, or such as have their origin during delivery, are by no means rare, but that the great majority of cases originate in the first three years of life. A later occurrence of the disease is less frequent, but it may appear even after the tenth year in a quite typical manner.

The acute *initial stage* may be lacking, the paralysis may develop without prodromal symptoms in the form of an apoplectiform attack, or in rarer cases, progress gradually to its full height. In the great majority of cases, however, the disease is ushered in by the above mentioned general symptoms —fever, vomiting, coma, and convulsions; the latter may be unilateral from the beginning. The initial stage lasts usually only a few days, but in rare cases, it may extend over several weeks. After the termination of the acute attack, the paralysis has usually developed to its full extent; but sometimes it happens, that the first attack leaves only slight disturbances, which, in the course of further convulsive seizures (after weeks or even months) by single steps, as it were, develops into a full-fledged hemiplegia.

The *paralysis* mostly *affects* the arm, the leg, and the face; but the *face* is as a rule involved only to a slight degree, and may even become again entirely normal in cases of old standing; the paresis of the face may be hardly recognizable during rest and in full innervation; but when the patient begins to smile or to cry, one recognizes, even in the lighter cases, the retarded onset or the too early cessation of the innervation on the paralyzed side (*Freud*). The ocular branch of the facial is frequently involved. The involuntary (mimic) innervation is far more frequently impaired than in adults (*König*). Crossed facial paresis may be simulated on the sound side, when the paralyzed side of the face is spastically innervated, whereby the corner of the mouth upon the affected side is drawn upwards and the affected eyelid is narrowed.

Of the other cerebral nerves, the *hypoglossal* is frequently affected; the tongue then shows a deviation towards the affected side; in such cases the facial is almost always also affected. Occasionally a paresis of the pterygoidei takes place also; in such a case, when the mouth is opened, marked lateral deviation of the lower jaw towards the hemiplegic side occurs (*König*).

Disturbances of the muscles of the eye, pareses of the oculomotor, are rare. Pupillary differences and reflex pupillary rigidity may point to syphilis as a causative factor (König, *Ossipow*); *hemianopsia* has been frequently observed (first by *Freud*); in younger children it can be found only after a very careful examination "through the indifference with regard to objects in the lost half of the visual field, and through the expression of helplessness and despair, if one places things, which were before seen and longed for, into the dark half of the visual field."

Motor *aphasia* occurs in paralyses of the right or the left side, is, however, not very frequent and usually does not last long.

Of the *extremities*, the arm is usually more seriously affected than the leg; and the hand, as a rule, most seriously. The leg also mostly regains its power more completely, so that in the older cases the impression of a monoplegic paralysis of the arm alone is sometimes gained. The paralysis is mostly restricted to single groups of muscles; *Lewandowsky* points out, that in contradistinction to the hemiplegias in adults, individual agonists and antagonists, for instance the flexors and extensors, retain their functional power always, while others, especially the rotators of the upper and lower extremities as well as the pronators and supinators, are paralyzed in pairs. The initial flaccid paralysis soon assumes the character of a spastic one, and in connection with it, *the exaggeration of the deep reflexes*, typical for the entire later course of the disease, appear, which, even in the most far reaching recovery of the motor functions, remain as a continual sign of unilateral cerebral paralysis. The cutaneous reflexes are mostly active. Frequently though not always, the *Babinski* toe phenomenon is present, extension of the great toe, when the sole or the inner side of the leg proper is stroked (*Oppenheim*).

Synchronously with the increase of the reflexes, the most significant symptoms of the disease, *rigidity* and *contracture*, appear; they are usually most pronounced in single groups of muscles, especially in the flexors and pronators of the arm, and in the flexors of the leg. Through this, in many cases, typical fixed positions are produced; the leg is constantly held in a slightly flexed position and rotated somewhat inward, the foot in spastic pes equinus position, the great toe extended dorsally, while the arm, pressed tightly to the body, is flexed at the elbow and thus held in an almost rectangular position (cf. Fig. 190); the hand may be fixed in a flexed or an overextended position; the thumb is turned inside the closed fist, sometimes, however, it is in a position of extension. In old cases, a passive overcoming of the spasms is frequently impossible; it may be that the origin of these fixed contractures is partly the consequence of a lack in conformity in the growth of the bones and of the muscles (*Lewandowsky*).

Besides this continual spastic state, which may be of the most varying degree and may even be lacking altogether, frequently the spastic innervation in voluntary movements is especially remarkable. These *intention spasms* hinder every action or render it difficult; the more intense the exertion of the will, the stronger the inhibition; the patients have constantly to subdue internal resistances, and their retarded and labored motions present a very characteristic picture. Frequently it can be seen, that there is hardly any reason to speak of actual paralysis, so that in some of these cases it seems more appropriate to use the designation hemitonia (*Bechterew*) or hemispasm instead of hemiplegia. In such cases, the spasms are less severe in passive movements than in active ones, they become insignificant or may even disappear entirely when the patient is lying down or sleeping.

There exists no direct relation between contracture and paralysis with regard to their intensity; frequently the spasticity is especially well defined in the leg, while the paralysis is localized principally in the arm; the hand, most markedly paralyzed, may be almost free from hypertonia, while the musculature of the shoulder may at the same time exhibit a condition of rigidity.

Abnormal passive motility is sometimes present in the joints of the hand and the fingers, so that the latter may be easily over-extended.

Because of the spastic fixation of the leg, the *gait* is usually very characteristic. The leg is not brought forward directly, but by a circular swing in the hip (circumduction); it drags slightly and the foot exhibits pronounced walking upon the toes. Later on the gait may become more normal, so that only a slight stiffness and helplessness remain, which, however, may become very perceptible, when the patient tries to stand on the affected leg alone, to jump, or to raise himself on the toes. It deserves to be mentioned, moreover, that the sound leg very frequently appears slightly spastic, if a thorough examination be made, and it may even happen, that later on with the gradual or attack-like progression of the cerebral process, disturbances in both legs may occupy the clinical foreground; these forms are then transitions to the diplegic ones.

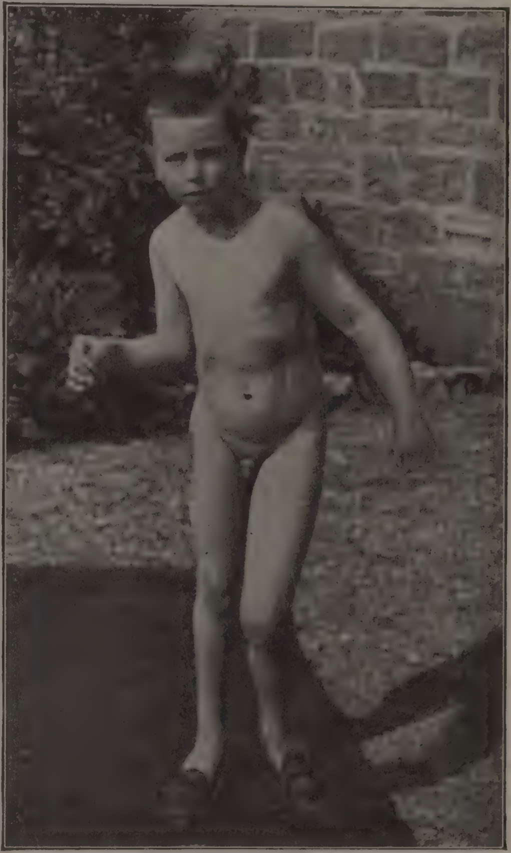

FIG. 190.—Infantile spastic hemiplegia of the right side. Contracture of the right arm, athetosis of the right hand, spasms and reflex augmentations in the left leg also, epilepsy, imbecility. (Observation at the children's clinic in Heidelberg.)

Disturbances of *sensation* are rare. The disturbance of the stereognostic tactile faculty, which has sometimes been noted, may find its explanation in the difficulty the patient experiences in feeling, because of the athetosis, or also in the fact, that, as the initial stage of the disease began in early life, the tactile memory pictures of the paralyzed hand had not been acquired at all (*Oppenheim*).

To the most important symptoms belong the *post-hemiplegic motor disturbances*. Of the spastic character of the volitional movements, we have already spoken; frequently it is accompanied by a certain degree of ataxia; tremor, also, and choreic twitchings may interfere with voluntary movement. *Associated movements* on the paralyzed side belong to the most frequent and typical phenomena of cerebral hemiplegia. As a rule, the paralyzed arm is raised to a more or less horizontal position as soon as the sound arm performs any kind of work; especially in walking or running the paralyzed arm may be seen to rise up like a wing and move to and fro like an oar. But the associated movements may be found in even much greater detail; the paretic hand may imitate all the motions of the sound one; also, associated movements of the sound side in intended movements of the paretic side of the body are frequently very clearly observed, and in the sound hand, the associated movement may surpass even in intensity the intended movement of the paralyzed one. Quite frequently, the patient tries his utmost to suppress or at least to diminish the associated movement, but without success.

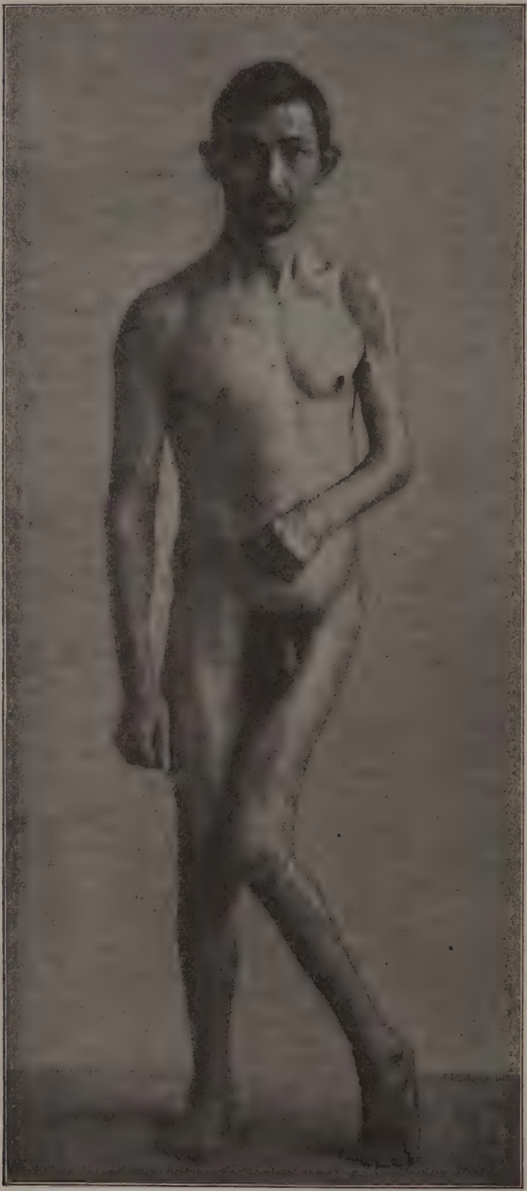

FIG. 191.—Infantile cerebral paralysis of the left side (acquired in the fourth year of life). The left arm, and less so, the left leg show a retardation in growth of a high degree. (*From Heinr. Curschmann.*)

The post-hemiplegic spontaneous movements, known as *chorea* and *athetosis*, are very striking. The involuntary, jerky motions of chorea affect usually the whole extremity, even the shoulder girdle, and may extend also to the face, while the slower, more rhythmic, stretching, bending and contracting movements of athetosis mostly affect the hands and feet; in the face, frequent grimacing may be regarded as an analogous phenomenon. These two types of motions, which likewise may be accompanied by lightning-like contractions (myoclonic movements *König*) distinguish the later stages of infantile cerebral paralysis; they may be found in about a third of all cases, and may be regarded to a certain degree, to bear a reverse relation to the amount of the paralysis; the more the paralysis and the spasms vanish, the more extensive and intense the spontaneous movements may become, in some cases, making the use of the limbs quite impossible. They cease, as a rule, entirely during sleep, with the exception of very severe cases of athetosis; on the other hand, they are very pronounced upon strong exertion of the will and in psychic excitement. Especially those paralyses, which begin in the later infantile age, are predisposed to chorea and athetosis. The spontaneous movements are usually strongest in the arm, and one meets, not infrequently, the combination of spastic paralysis in the leg and athetosis and chorea in the arm.

Trophic disturbances in the limbs affected by hemiplegia are frequent, especially a retardation in growth of the extremities affecting their length and thickness (cf. Fig. 191), which latter may be partly caused by an osteoporotic condition of the bones, as the X-ray pictures show (*Kellner*, *v. Rutkowski*).

A more thorough examination will mostly show, that the inhibition in growth does not affect the whole limb equally. Hypoplasia shows a predilection for the cases that start in early childhood, and is most clearly defined in the second period of childhood and at the time of puberty; it develops quite independently of the degree of paralysis and may, in some rare cases, become the most conspicuous symptom.

The *musculature* of the paralyzed side of the body shows occasionally an atrophy, surpassing by far the atrophy caused by simple inactivity. In athetosis, hypertrophy of the muscles may sometimes be observed. The other trophic disturbances (of the breast, the testicles, abnormal development of the pubic hair, precocious local *canities*, etc.) are of minor interest. A sub-normal temperature and a bluish-red discoloration of the paralyzed extremities are observed, but mostly in a very moderate degree.

The *skull* frequently shows asymmetry, flattening of the parietal bone, etc. On the other hand, microcephaly of a higher degree is rarely ever found in the hemiplegic forms.

Of decisive importance for the later fate of the patients, is the very frequent complication of the disease with epilepsy and idiocy.

More than half of the patients become *epileptics*. Usually the convulsions, which accompany the initial stage of the disease, are followed by an interval of weeks and months, and even, not very rarely, of many years, in which no attack occurs; then without any warning epileptic convulsions, recurring at more or less frequent intervals, make their appearance, and may lead to the status epilepticus. Even the least serious cases, in which the paralysis has left only insignificant traces, are by no means secure from this serious complication.

The attacks can be distinguished from real epilepsy, at least in the first years after their appearance, by the frequently unilateral onset or course and by their lesser severity; they are, furthermore, mostly preceded by an aura and show only rarely the initial shrill cry; involuntary evacuation and biting of the tongue occur only rarely; loss of consciousness may be lacking entirely, or be of short duration, and the after stage of the disease is usually lacking. Psychic equivalents have been noticed only in isolated cases (for instance, by *Heubner*). According to *Bourneville*, the period of severe epileptic attacks, lasting usually from 10 to 15 years, is followed later by an alleviation in the epilepsy, and it may disappear entirely between the 40th and 45th year.

Besides the epilepsy, *disturbance of intellect* is a sad adjunct of many cases of infantile hemiplegia. Only in a minority of cases do the mental faculties remain perfectly intact, and even in these a change in character may usually be noticed, irritability and a tendency to fits of anger and passionate acts; all degrees of disordered intellect may occur, from slight imbecility to the most severe idiocy, but the latter are more frequent in the diplegic forms of cerebral paralysis.

As to the disturbance of intellect, there is also no parallelism with the seriousness of the paralysis; there can be hardly any doubt, that there are cases of pure idiocy, which have to be regarded as identical with those in infantile cerebral paralysis, but do not exhibit any symptoms of paralysis, because the process of the disease has spared the motor centers, and has played its part only in the so-called "silent" portions of the brain. Such cases are designated, after *Freud*, as "cerebral paralyses without paralysis." Cases of deaf mutism also may belong to this category (*Schultze*, *Heubner*).

PECULIAR TYPES OF THE DISEASE

1. *Strümpell's encephalitis acuta.*—Under this designation must be classed those acquired cases, in which a "massed" appearance, for instance in a whole family, or the typical febrile onset accompanied by convulsions, coma, vomiting, etc., imparts the impression of an acute infectious disease. In all other respects the course of the disease does not deviate from the one mentioned above.

2. *Choreic paresis* (*Freud and Rie*).—This name denotes those disease pictures, in which the hemi-chorea was not preceded by a spastic paralytic stage, so that this seems to have been skipped. These cases have the peculiarity, that they mostly occur in children somewhat older than infants (more than 3 years), that the development of the disease is, as a rule, insidious

and that epilepsy, atrophy, aphasia, and disturbances of the intellect in a higher degree are almost always lacking. In respect to the ætiology of these cases acute infections and psychic trauma (terror) play an especial rôle.

Diplegia Spastica Infantilis; the Cerebral Diplegias of Children

Part of the cerebral diplegias represent nothing more than a duplication of the hemiplegic paralysis. With *Freud* we designate such cases *bilateral spastic hemiplegia.* This affection exhibits the same characteristics as unilateral paralysis, the usually greater involvement of the arm, the contracture positions, etc. The mind is frequently severely affected. Paralyses of the cerebral nerves may become of special importance in these cases, of which more will be said in the chapter on pseudobulbar paralysis.

The most important forms of cerebral diplegia however are represented by cases, commonly known in the literature as *Little's disease*, which we designate with *Freud* as *general rigidity* or *paraplegic rigidity*, according to whether the whole body or only the legs are affected. *These cases are distinguished especially, in that the paralysis plays a more or less secondary rôle and may even be entirely lacking, while the stiffness, the rigidity of the musculature, dominates the entire disease picture, furthermore in that the legs are predominantly or exclusively affected, then again through the pronounced regressive tendency of the disease phenomena in many cases and through their ætiology, which in the great majority of cases can be proved to be of prenatal origin or dependent upon injuries during* birth. Asphyxia and difficult labor are mostly found in the anamnesis of general rigidity, premature birth on the contrary usually in that of paraplegic rigidity (*Feer, Freud*).

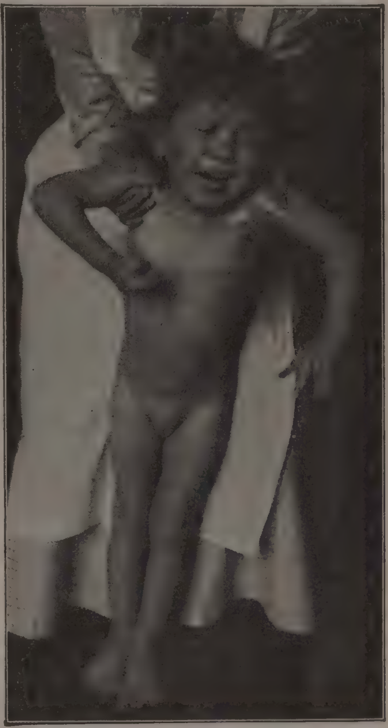

FIG. 192.—General rigidity (Little's disease), athetosis, idiocy. (Author's observation at the children's clinic, Heidelberg.)

Serious cases of general rigidity attract the attention of the parents even during the period of lactation; during the bathing the infants appear stiff and unmovable like a log of wood, and it is difficult to dress them. The legs can hardly be separated; it is impossible to place the children in sitting position, as at each attempt of this kind the whole trunk is stiffened like a board. Frequently less serious cases, even when their prenatal origin can not be doubted, are not discovered till an attempt is made to teach the children to walk, which is frustrated by the stiffness of the legs. Many children do not learn to walk at all or only at a very late period. If they are placed upon their feet a very characteristic picture appears. The thighs are rotated inward, the knees are pressed together because of spasm in the adductors, which, as a rule, is quite pronounced, and the feet touch the ground only with the extreme tips of the toes. When laboriously trying to walk, the knees constantly rub one another and the legs cross each other constantly. The child does not walk, it winds itself, as it were, around its own axis (Fig. 193). In cases, where the spasms are less marked or show a certain retrogression in later age, the children can walk, it is true, but have constantly to overcome an internal resistance. In their laborious and slow stepping out, they make the impression of one who moves forward in a slimy substance or wades in mud. Walking upon the toes, at the same time, is constantly retained. When lying down or getting up, the legs are frequently moved in unison as if they were welded together. In a sitting position the legs, because of the extension spasms, protrude straight into the air. The big toes as a rule are stretched outwards and upwards.

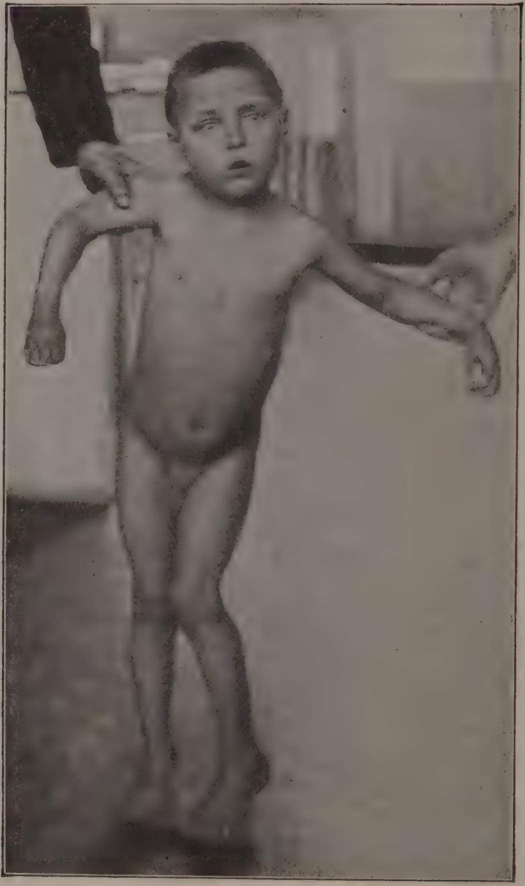

FIG. 193.—Gait in paraplegic rigidity (Little's disease). Arms entirely free. Premature delivery. Retarded psychic development. Cryptorchidism. (Author's observation at the children's clinic, Heidelberg.)

The symptoms of rigidity are always less pronounced in the arms; typical permanent contractures as in hemiplegia do not occur. When the face takes part in the rigidity, it assumes a mask-like, almost invariably uniform expression; in crying the facial expression appears forced, sometimes in a quite odd way resembling the usual expression of the face when smiling (perverse mimicry, *Freud*). Spasms of the musculature concerned in swallowing may impede deglutition. Choreic and typical rhythmic athetotic motions are by no means so frequent as in hemiplegia; it is frequently seen however that the hands in motion assume peculiar athetoid positions (cf. Fig. 192). Tremor and ataxic phenomena may also occur in these cases. The deep reflexes are always increased, but very frequently, when tapping the tendons no muscle contraction at all can be observed because of the rigidity. According to my experience the crossed adductor reflex is very frequently seen to appear (upon tapping the quadriceps tendon contraction of the contralateral adductors). The *Babinski* toe reflex is usually present. Lately *Brudzinski* has called attention to peculiar contralateral reflexes; if one leg is flexed passively, the other is automatically extended or possibly also flexed.

In some cases the spasms may become more or less latent and manifest themselves only upon the stronger volitional motions or when passive movements are executed in an irregular, jerky manner. Sometimes any unexpected touch, even acoustic irritations cause a sudden start of the infant, followed by an intense spastic extension of its whole body.

Concerning the *cerebral nerves*, it should be mentioned that *strabismus* is to be found very frequently, especially in paraplegic rigidity; atrophy of the optic nerve is less frequent, though, if a thorough examination be made, not so rare as is assumed as a rule. Pupillary differences occur. *Kōnig* has pointed out the occurrence of so-called "changing pupils" ("springende Pupillen"), namely that in different examinations now the right and now the left pupil is more contracted. Nystagmus is frequently observed, also nystagmus verticalis (my own observation). Dysarthria and bradylalia are not uncommon.

The sensibility does not show any changes (except in cases of idiocy, when it is benumbed).

Hypoplasias, as occur in hemiplegias, are scarcely ever seen, but occasionally an arrested development of the entire body can be observed. Muscular atrophies are pronounced only where the paralyses reach very high degrees (paraplegic paralysis); but hypertrophies of single or almost all groups of muscles have been observed as a consequence of the hypertonia (*Ibrahim*, *Finkelstein*). We may mention here also the high location of the patella with the lengthening of the ligamentum patellæ, which has been described by *Schulthess* and *Joachimsthal* and which is observed best when the knee is flexed into an acute angle.

Convulsions are very frequent in all forms of cerebral diplegias; in common with *Freud* it is proper to distinguish between early convulsions appearing in the first days after birth and being frequently the consequence of a traumatism during delivery, and the spasms that appear later on (late convulsions) which may be regarded as being of an epileptic character. The latter, however, are much less frequent in diplegias than in hemiplegias, and in paraplegic rigidity they are even quite uncommon.

Defects of intellect play a very important part in diplegic cerebral paralyses. A great majority of these defects are the result of grave malformations of the brain, prenatal disturbances in development, and these cases usually show most marked idiocy. With comparative frequency these patients present the symptoms of general rigidity; not rarely a microcephalic cranium is found in these cases, which frequently is noted already at the time of birth. Other malformations are rather rarely present at the same time. In the cases due to traumatisms during delivery and in the so-called bilateral spastic hemiplegias the mental faculties are often badly impaired. But cases with an almost or an entirely normal intellect occur also.

As to the **course** of the so-called *Little's* disease it has already been mentioned, that it is usually regressive; it may happen that nothing is left of the general rigidity but a stiffness in the legs and that the paraplegic rigidity may almost entirely disappear in the course of years; still the faculties of gait and speech are often impaired for a long time afterwards. The mental faculties, as a rule, do not show such a tendency towards restitution.

Pathological Physiology.—The real nature of the rigidity has not been explained as yet uniformly and satisfactorily. Most authors accept the theory, that the loss of the inhibitive influences, which the cerebrum exerts upon the spinal cord, is the causative factor. The loss of the pyramidal tracts (by atrophy or non-development) may play a part, but certainly is not the paramount or exclusive causative factor; other well known or even unknown connecting pathways between cerebrum and spinal cord may be concerned therein. *Freud* explains the existence of rigidity without paralysis by the fact, that the cerebral lesion is only incomplete or superficial, so that the influence of the cerebrum on the innervation of the spinal cord is merely weakened but not lost. According to his deductions the cortical tract possesses a by far greater influence upon the upper extremities than upon the lower. "If the cortical influence be entirely lost this fact must be made more noticeable in the arms, by a higher degree of paralysis and contracture; if, however, it is only weakened, it may still be of some import for the innervation of the arms, so that these are better protected from paralysis and contractures than the legs."

It should be mentioned here also, that *Rolly* on the strength of four uniform findings in personally performed autopsies has tried to fix a typical pathologico-anatomical basis for the cases of pure rigidity without a vestige of paralysis, which he sees in an absolute absence of macroscopical changes combined with microscopically provable diffuse proliferations of the glia tissue and of the blood vessels (in the brain and spinal cord); he suggests for this type of disease the designation of *general congenital muscular rigidity*.

PECULIAR SYMPTOM COMPLEXES

1. *General chorea and bilateral athetosis.*—Part of these cases correspond to a duplication of the choreic paresis mentioned above; but the paralytic phenomena are usually more pronounced, than in the hemiplegic form. General chorea, in which the musculature of the trunk and the face usually participate, causes serious disease pictures. At the same time the intellect is frequently much less impaired than would seem likely, considering the inhibition that exists in freedom of movement and power of speech. Hypertonia is mostly restricted and slight, or even an abnormal flaccidity of the muscles may be present.

Bilateral athetosis (*athétose double*) shows pronounced analogies with bilateral chorea. It appears as a concomitant of serious spastic congenital diplegias and is characterized, according to *Lewandowsky*, by a kind of generalized but not at all identical associated movements, which may be excited by even quite slight psychical emotion. The spontaneous movements disappear during sleep or rest; as soon however, as the patients think, that they are being observed, a wild grimacing of the face and a constantly changing motion of all the limbs commences, which assume athetoid postures of all kinds in a way, that each motion excites other still more odd associated movements. The gait also is not simply spastic, but oddly distorted. The cerebral nerves may exhibit phenomena of pseudo-bulbar paralysis.

2. *Pseudo-bulbar paralysis*, which has been described minutely, especially by *Oppenheim and Peritz*, is a symptom complex, which in all forms of infantile diplegia, in the congenital with general rigidity as well as in the acquired forms with bilateral hemiplegia and even in those types of the disease, which develop chronically, may complicate the disease picture and may sometimes, because of the serious disturbances that it occasions, step into the foreground. The features have something rigid, masklike. In cases, which show a higher degree of paresis, frequent bursts of emotion, laughing and crying may accompany a perfectly normal innervation of facial expression (*Peritz*); in the more spastic forms facial expression may be entirely lacking, the face looks then, as if carved of wood; or the right measure of the motions may be lacking and instead of smiling the face assumes the distorted expression of the risus sardonicus. Speech is made difficult, the voice sounds nasal, even when the volitional raising of the soft palate is retained, or scanning, slow; it may also be entirely lacking or aphonic. Chewing and swallowing, the finer movements of the lips, cheeks, and tongue, are disturbed, and sometimes, the taking of nourishment is made difficult. But it is very remarkable, that it is principally the volitional movements, that can not be executed, while the involuntary innervation is

co-affected only in the most serious degrees of the disease, especially in the spastic forms. The children can not purse the lips, can not whistle, blow out the cheeks, stretch out the tongue, they can not spit out; sucking and swallowing, on the contrary, are relatively well performed. Sometimes they must use their fingers to push the bits of food back into the region of the more automatically working parts of the oral cavity, whence the further process of deglutition is performed in a normal manner. Obsessional laughing and crying are rare in children. On the other hand the musculature of the nape of the neck is quite frequently paretic, so that the head can not be held in a straight position. Besides these severe forms, there appear all possible gradations to the very slightest disturbances, which manifest themselves only by changes in speech and by keeping the mouth open.

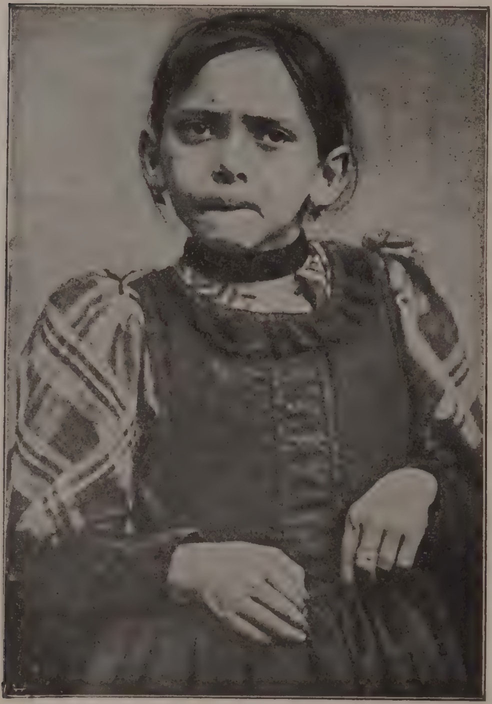

FIG. 194.—Cerebral diplegia with very pronounced spastic pseudo-bulbar paralysis and athetoid position of both hands. The mother showed the same disease picture. (A case of Oppenheim's, from his Lehrbuch der Nervenkrankheiten Auflage.)

Of special interest are two symptoms described by *Oppenheim*, which are frequently found in such children—the *abnormal timorousness;* the sudden start at the slightest noise; and the so-called *eating reflex* (the appearance of a large number of rhythmical chewing, sucking and swallowing movements upon stroking the lips or the tongue).

The explanation of these symptoms, as well as the highly interesting pathological physiology of the entire symptom-complex of infantile pseudobulbar paralysis, can not be treated here more minutely. Concerning pathological anatomy and ætiology, everything, that was said above regarding infantile cerebral paralysis, is valid.

3. *Infantile spastic spinal paralysis, tabes spasmodique.* Formerly it was customary to designate thus a number of cases, which, in view of our present knowledge, we are now inclined to regard as being surely of cerebral origin. It concerns that group of diseases, which we have designated above as paraplegic rigidity. We have mentioned that strabismus, disturbances of speech, defects of intellect, epilepsy, etc., may be found in such cases. For

cases of this character the cerebral origin of the disease is at once clear; on the other hand, in children who show only a spastic paraparesis of the legs,[1] or of all four extremities, because of clinical considerations, the possibility of a purely spinal origin of the disturbance in motility, can hardly be denied. As a matter of fact, *Déjérine* has even succeeded in finding in two such cases, a focal injury (syphilis?) in the cervical portion of the spinal cord, with secondary degeneration of the pyramidal tracts as a primary basis for the affection. Because of the fact, that such cases occur frequently in premature delivery, some authors (*van Gehuchten*, *Brissaud*) have expressed their opinion, that the rigidity is dependent upon the immature development of the pyramidal tracts and their medullary sheaths, in this stage of fetal life; and the later improvement of the disease is explained by the further growth of these tracts. This line of thought seems attractive; but it ought not to be limited to the pyramidal tracts and the ætiological factor of premature births alone, as in such a case, many facts and considerations would serve to refute it. The fact, however, that because of intrauterine injuries, combined perhaps with the factor of premature birth, the development of all connecting tracts between cerebrum and spinal cord may be arrested or retarded, a defect which, in later times, may improve, more or less, could certainly be of some advantage for the explanation of many cases of this category, especially as thus far not sufficient post-mortem material is known which would refute this theory.

4. *The familial forms of cerebral diaplegia* as well as *amaurotic idiocy* will be treated in other chapters of this book.

Diagnosis.—Well defined cases of infantile cerebral paralysis, hemiplegic as well as diplegic, are usually easily recognized; on the other hand, difficulties may arise if the typical symptoms of paralysis and rigidity have disappeared to such an extent, that only traces are left, and other symptoms step into the foreground. Because of these possibilities, it is necessary to examine every obstinate case of *chorea*, especially hemichorea, as to spastic residues and unilateral exaggerations of reflexes. The recognition, especially, of choreic paresis may be sometimes impossible in the initial stage. In the same way all cases of *epilepsy* and *idiocy* must be examined for residues of rigidity, *i. e.* the presence of athetotic or choreic disturbances, and exaggeration of reflexes, if the character of the disease is to be judged correctly. From the peripherally induced paralyses (*poliomyelitis*, *obstetrical paralysis*), which may show superficial similarities, cerebral paralysis is easily distinguished by its spastic character, the exaggerated reflexes, the slighter degree of atrophy, the mental disturbances, the epilepsy, and the choreic and athetotic movements. If there still be any doubt, the retained excitability of the paralyzed muscles to the faradic current, will exclude any idea of peripheral paralysis.

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[1] Some authors classify only these cases as *Little's disease*.

In the acute initial stage, the character of the disease cannot be recognized before the appearance of paralysis. The inclination in every case is rather to think of a *meningitis.* Tubercular meningitis, moreover, may occasionally set in with a hemiparesis or a hemichorea, so that in these cases, also, only a thorough and painstaking study of the further course of the disease will prevent errors in the diagnosis.

Cerebral tumor may pursue a similar course to cerebral hemiplegia; when other symptoms, choked disc, etc., are lacking, only further observation of the disease will insure a definite diagnosis. The same is true of *cerebral syphilis,* which should be thought of, however, in cases with pupillary differences and pupillary reflex rigidity.

Differential diagnosis, relative to *multiple sclerosis,* will have to be taken into consideration in those cases, where nystagmus, intention tremor, and bradylalia are combined with paraspastic conditions. The anamnesis in such cases will eventually decide for the diagnosis of cerebral diplegia. The familial cases of diplegia, especially, may lead to difficult differential diagnostic problems. But we must remember, that multiple sclerosis in children is very rare indeed, and possibly never occurs in a well-pronounced form.

From *Friedreich's ataxia,* the cases of cerebral diplegia, in which ataxic symptoms, bradylalia, and nystagmus appear, are distinguished by the spastic phenomena and the increase in the reflexes. The familial cases of spastic diplegia are naturally recognizable as such only, when other children of the same parents have already been affected. In cases with uncommon concomitant symptoms, this must be taken into consideration.

That *hydrocephalus* may lead to clinical pictures, which bear a great similarity to those of paraplegia, and even of general rigidity, is well known, especially through *Ganghofner.* In infants, the cranial size indicates at once the correct diagnosis; in children of a more advanced age, a comparatively rapid growth in the size of the skull, and the appearance of the disturbances in the optic nerve will lead to the diagnosis "hydrocephalus."

Diffuse sclerosis of the brain and *Westphal's pseudosclerosis* may be distinguished from cerebral diplegias by their more insidious onset and the progressive development of the symptoms, furthermore, by the more advanced age of the affected children, at which acquired cerebral hemiplegias only rarely appear, and by some other clinical peculiarities (apoplectiform attacks, etc.).

The differential diagnosis between the individual forms, especially of the diplegias, is to be made from what we have remarked under symptomatology. Pure forms of the disease are rarely found, mostly mixed forms. Frequently only when the initial stage of the acquired paralyses is well defined, can the anamnesis decide, whether the case is a prenatal, an acquired case, or one that originated intra partum. Microcephaly in a higher degree points, as a rule, to a prenatal origin (*Ibrahim*).

As there is no constant relation between the clinical pictures and the pathologic-anatomical findings in the brain, a sure recognition of the latter is out of question; the idea that a diagnosis of porencephalia, or cerebral atrophy, etc., may be made in vivo, will have to be abandoned. Adenoma sebaceum of the skin as well as congenital tumors of the kidneys or the heart may, according to *Vogt*, be considered in a diagnosis of tuberous cerebral sclerosis.

Finally, it must be pointed out how difficult it may be, to recognize with certainty in the early period of lactation slighter cases of general or paraplegic rigidity, as in the young infant, physiologically, the musculature shows a considerable degree of stiffness. Besides, it is to be considered, that in this tender age, as reports of later years have made increasingly certain, hypertonias of the musculature and permanent spasms may occur in nursing infants as concomitants of acute and especially of chronic disorders in nutrition, and may last for weeks and even months to disappear entirely with improvement in the conditions of nutrition. These spasms may easily mislead the inexperienced, and make it difficult for the experienced practitioner to make a certain diagnosis of disturbances situated in the cerebrum. The other accompanying symptoms of *Little's* disease, especially idiocy, also a clearly pronounced increase of the reflexes will make the diagnosis more certain. According to my experience the detection of a pronounced crossed adductor reflex, as well as the discovery of a premature closure of the fontanelles may be of value.

Prognosis.—The course of many hemiplegias and of numerous cases of general and paraplegic rigidity is a regressive one. In the course of years the motor disturbances improve, and the children of the last group (affected with paraplegic rigidity) finally learn to walk after reaching the 8th to the 10th year. One should not be too sure, however, of a far-reaching spontaneous cure. Of the greatest importance for the prognosis of all forms of infantile cerebral paralysis is the amount of the existing *mental* faculties, as the therapeutical success depends quite considerably upon the co-operation of the patient, and the chances of recovery upon the patient's intellectual plane not being too low.

Chorea and athetosis, which frequently in hemiplegia take the place of paralysis, do not make the functional use of the arm any easier, though it is really astonishing, how dexterously such patients sometimes use their hands, notwithstanding the almost uninterrupted spreading movements of the fingers, how they hold the tools securely and learn to handle them; nevertheless, the great majority of the patients remain permanently incapacitated for work. Even in the lightest cases the prognosis is darkened by the fact, that even after a lapse of many years one is never secure from grave complication with epilepsy, which for the future life of the patient has as much serious importance as genuine epilepsy.

The duration of life is, as a rule, not shortened by the cerebral paralyses nor by their consequences.

Treatment.—Prophylaxis is possible only in so far as its aim is an artificial acceleration of protracted labors. From this viewpoint *Sachs* has advised in such cases the early use of the forceps.

A causal therapy in the initial stage has been tried in recent years in severe meningeal hemorrhages caused by obstetric traumatisms. Lately, *Seitz* has studied more closely the symptomatology of these conditions and has reported an operative experiment (opening the parietal bone, and washing away the coagulated blood). *Cushing* in two such cases, effected a cure. Possibly further attempts in this direction will prove successful.

Besides these cases, causal therapy is possible only in cerebral syphilis. Where such a suspicion seems justified, under all circumstances an energetic anti-syphilitic treatment is indicated.

In the acquired forms, the initial stage will have to be treated by the utmost rest, an ice cap or cooling tube on the head, and derivatives, as calomel, internally. For the early convulsions of the congenital disease, hydrate of chloral had best be used.

The treatment of the paralyses when continued with steadfastness and patience may frequently show good results.

In the *hemiplegic forms* one will make use of electricity: of the faradic current for the paralyzed groups of muscles and of the anode of the galvanic current in cases of spastic contractures. Cautious massage, warm baths, and above all active and passive movements may prevent the formation of contractures. Purposive active exercises may be of great value, but they are dependent upon considerable use of the will power of the patient, hence this method of treatment can only be applied to children, who have passed the tender age and whose intellect did not suffer to too great a degree. The tendency to associated movements may be utilized therapeutically by using also the sound hand in performing the exercises (*Hans Curschmann*). Treatment by muscle exercise is of especial value in those cases, in which by *tenotomies* and reductions the contracture positions have been removed.

Orthopedics may also in other respects improve the condition of the patients. Through *transplantation of tendons* a weakening of the spastic and a strengthening of the paretic groups of muscles may be brought about by transferring parts of the hypertonic muscles to the areas, where the function of the muscles is impaired. Of especial importance are the reports of *Wittek* and *J. Fränkel*, which show, that the occurrence of chorea in the limb, that was subjected to a tendon operation, was directly prevented, because of the change effected in the periphery. The importance of this can not be overemphasized, if one considers, that chorea and athetosis usually are very little susceptible to treatment. *Sachs* reports improvements and even permanent cures of athetoid movements through wearing an apparatus of splints for several months, which made these movements impossible.

For the **treatment** of *epilepsy* we have no other means at our disposal than those used in the treatment of the genuine form of this disease; a non-irritating diet with little salt and no alcohol and the bromide preparations. It has been frequently attempted to influence the paroxysmal attacks by surgical interference such as trephining the cranium and excision of meningeal scars or evacuation of cysts. The successes have thus far not been very numerous nor permanent; notwithstanding it is advisable to try surgical treatment along these lines, whenever there are grounds for the assumption of a localized focus of disease.

Little's disease, especially paraplegic rigidity, offers many points of vantage for treatment. The hypertonias are, as experience proves, favorably influenced by protracted warm baths. *Heubner* recommends repeating a regular bath treatment, lasting several weeks, several times a year. "One begins with 98 degrees ten to fifteen minutes duration and progresses by degrees to 100-101 and even more, lengthens also the duration of the bath and makes the patient perspire somewhat afterwards. The baths are to be taken every other day. Children of well to do parents are best sent to summer resorts as Wildbad Gastein, Teplitz, and similar localities."

Massage, especially in the warm bath, and very cautious passive movements may tone down the rigidity of the muscles even further. By massage one tries to strengthen the abductors and extensors and by tapotement of the tendon ends (Hoffa) to lower the spasms in the adductors and flexors.

The principal consideration with such children is to get them upon their legs, to make them capable of independent locomotion. If the rigidity is very pronounced, the goal can be reached only by radical orthopedic measures, which in such cases are, indeed, of very great value. It does not suffice to remove the principal contractures by tenotomies of the adductors and of the Achilles tendon; to reach a satisfactory success here, at the same time also transplantations and shortenings of tendons, in different ways according to the individual case, have to be performed. The children must then be placed for a long time in a plaster bandage in a straddling position. An appropriate after-treatment with active and passive movements and systematic walking exercises, resp. at first in orthopedic apparatus will finally bring about the desired result; it is remarkable, that by the transplantation of tendons, as numerous experiences show, the spasms too, as a rule disappear entirely (*Vulpius*). As has been said before, the whole treatment is only of value when the intellect of the little patient has not suffered too greatly.

Speech also in many cases presents a fruitful field for therapeutic endeavors, since it is frequently in such children markedly deficient only, because it is taken for granted, that a severe idiocy exists and therefore no well-defined attempt is made to be of service in this regard. Frequently the patients need a special pedagogic treatment, while in cases of more serious idiocy placing them in proper institutions is to be recommended.

2. CHRONIC HYDROCEPHALUS OF CHILDREN

Chronic hydrocephalus of children is not a disease concept strictly demarcated ætiologically and pathogenetically. We rather include under this designation a number of conditions, *which all have one symptom in common, an increase in the cerebral fluid.* Without doubt an ætiological division of these different diseases would be highly desirable, but practically it is not feasible because of the obscurity, which, as a rule, covers the pathogenesis of each individual case (at least during life). On the other hand, the symptoms excited directly or indirectly by the collection of the fluid masses dominate the disease picture (and the pathologic-anatomic changes) to such an extent, that because of this one symptom alone a comprehensive discussion under one heading may be regarded as justified.

If we therefore start from this point of view, it will be easily understood, why we do not think it appropriate to follow the common usage of treating separately hydrocephalus congenitus and hydrocephalus acquisitus, as both these forms, as a rule, appear clinically absolutely similar and as in the individual case, even with the aid of lumbar puncture, no distinction can be made; indeed, the anamnesis even does not always lead to a certain conclusion. Special diagnostic and prognostic pointers for judging the individual case will occasionally be given. We regard a separation of the cases occurring in the first years of life from those in later years of greater importance, since the latter, because of the closing of the sutures and fontanelles, exhibit clinical pictures quite different from the former.

Before entering more minutely into the symptomatology we wish to mention a few types, which practically are of minor importance.

1. *Marantic hydrocephalus*, which is found occasionally as a pathologic-anatomical secondary finding in tuberculosis, nephritis and anemias, has usually a course entirely devoid of symptoms and may therefore be omitted in this discussion.

2. *Hydrocephalus e vacuo* occurs in inhibited development and shrinking of the brain caused exclusively by the misproportion between the skull cap and the cranial contents (*hydrancephaly*) and may combine with microcephaly; it too is devoid of any independent importance.

3. *Hydrocephalus externus* (*hygroma duræ matris*).—Thus an abnormal collection of fluid between the dura and pia is designated. Hydrocephalus e vacuo is to be named first in this connection. Whether a genuine hydrocephalus externus occurs in other cases is doubtful, but surely to be regarded as probable in view of some recent observations (*Léon d'Astros, Huguénin, v. Bókay*). It seems, that in these cases the development of the brain may be retarded through the primarily existing fluid mantle. But as a rule it is, as we know with certainty, a matter of consequent or concomitant phenomena of a pachymeningitis hemorrhagica interna, which ought not to be con-

founded with chronic hydrocephalus, if from no other reason than the inflammatory character of the fluid which may be localized in cyst-like intrameningeal cavities (hydrocephalus saccatus). Concerning symptomatology this form of hydrocephalus externus may exhibit clinical pictures quite similar to those of hydrocephalus internus, though the quantity of fluid is usually not so large.

Hydrocephalus Chronicus Internus

In this common form of hydrocephalus, quite a frequent disease of childhood, the fluid collects in the ventricles of the brain.

Pathological anatomy.—The lateral ventricles are, as a rule, most dilated, but the third and fourth ventricles also may participate to a more or less extensive degree and even the uppermost section of the spinal canal may be dilated. The enlargement of the lateral ventricles is usually symmetrical to a certain extent, and also the foramina Monroi are as a rule retained, frequently even abnormally wide (up to 5 cm. diameter). There are, however, also cases of unilateral effusion (hydrocephalus partialis). The quantity of the fluid may be increased but slightly, but it may also attain excessive degrees, especially in babies with a yielding skull cap. On the average it amounts to 1/2 to 1 liter, but quantities of 10 and 12 liters and even more have been reported. Of the physical and chemical composition of the fluid we shall speak later.

The changes in the substance of the brain are directly dependent upon the pressure of the fluid. The cerebral cortex at first is less affected. The medullary substance of the cerebrum as the corpus callosum and fornix are the first to suffer atrophy (*Anton*). But when the disease reaches higher degrees, the convolutions and the fissures are filled out; in fact, it may happen, that post mortem the entire cerebrum presents the appearance of two large tensely filled cysts, the walls of which in the most extreme cases are formed by a layer as thin as a sheet of paper, which can be identified as a remnant of the gray cerebral substance only after a painstaking histological examination. The central ganglia, too, not rarely are damaged and flattened by the effect of the pressure; the floor of the third ventricle may bulge out bladder-like and thereby markedly compress the chiasma of the optic nerves. The cerebellum by the effect of the pressure of the fluid may be pushed partly or entirely in the direction of the dilated vertebral canal (*Chiari*); in some cases it has been found compressed to the thinness of a leaflet.

When the hydrocephalus has its origin in fœtal life, serious inhibitions in the development of the entire brain may be the consequence. In such cases hydrocephalus may be combined with microcephaly. Non-development or arrested development of the pyramidal tracts may likewise be found; according to *Engel* it is principally a matter of retardation in the normal medullary sheathing of these tracts. But secondary degenerations occur also (*Schultze*).

The changes in the *ependyma* are neither constant nor typical. It appears sometimes thickened and granulated; inflammatory changes, too, have been found in the blood vessels of the subependymal tissues. The *plexus chorioidei* frequently exhibit changes of an inflammatory nature, connective-tissue thickenings and cystic formations.

The *meninges* frequently are perfectly normal. In a number of cases, however, turbidity and thickening, especially at the base of the brain, may be found. This applies especially to the acquired forms, which in the majority of cases may be regarded as final stages of a meningitis, which in all other respects has been cured.

In the more severe forms of the disease the *cranial bones* become atrophic through pressure; the diploe disappears. At the same time the bones themselves are, as a rule, considerably larger than the normal ones. Frequently one finds Wormian bones (intercalares). Of the behavior of the sutures and fontanelles we shall speak in the clinical discussion. At the base of the skull a premature synostosis may inhibit the growth of the bones.

The rest of the body does not show anything which could be brought into connection with the hydrocephalus. The only report to the contrary is that of *Czerny*, who in five cases found hypoplasia or non-development of the medullary substance of both *supra-renals* with macroscopically normal appearance. There is no consensus of opinion whether any importance should be attributed to this fact.

It should also be mentioned here that the congenital hydrocephalus is very often combined with malformations of all kinds. Of special frequency is the combination with spina bifida, a disease which pathogenetically exhibits a certain analogy to hydrocephalus. Of special interest is the fact that after a removal of a myelocele not infrequently a chronic hydrocephalus has appeared. Besides club foot, hare-lip, cleft palate, coloboma of the choroid (*Saemisch*), vitiligo (my own observation), albinism (*Oppenheim*) and other diseases have been found combined with hydrocephalus.

Clinical picture.—The more severe forms of chronic hydrocephalus *in the first or second year of life* exhibit usually the following symptom complex.

The monstrous dimensions of the head attract immediate attention; it forms a considerable part of the entire length of the body—a fifth and even more. Its circumference surpasses considerably that of the thorax and reaches to 60 and 70 cm.; through the thin, sparsely grown hair one sees even from afar the lines of blue veins meandering upward from the temples and from the root of the nose, which during crying spells may swell in bold relief like cords. The disproportion between cranium and face is surprising; the latter, small and daintily fashioned, broadens in the vicinity of the eyes and thereby approaches a triangular form, which because of the adjoining gigantic part of the cranium with its globular vaulting assumes a pear-like shape. This pear-like shape is equally pronounced whether one looks at the head from the front or from the side. The high protruding forehead, in which the tubera frontalia appear strongly arched, is divided almost to the root of the nose by the extended large fontanelle. There are as many centimeters from the border of the hair to the root of the nose as from here to the chin. The root of the nose itself is broad and the eyes widely separated. Laterally from these the temples exhibit strongly arched hemispherical protrusions; it becomes evident, that here the lateral fontanelles have opened again and exhibit a wide fissure. The ears stand obliquely from the head and the external auditory canal has changed its whole direction, is transformed into an almost horizontal slit. The sagittal and lambdal sutures have the appearance of wide canals, wide enough to put several fingers in. The marked tension of the fontanelles makes it impossible to recognize respiratory or pulsatory changes.

The eyes converge and are pushed downwards. The dilated, rigid pupils are partly covered by the lower eyelids. A large part of the sclera becomes visible between the upper eyelid and the iris, and the two white spots in the shapeless face shine uncannily. A slow nystagmus increases still more the restless impression.

The head looks like a motionless mass, to which the rest of the body is attached. A certain stiffness in the extremities may be observed at the first glance. The thumbs are turned in, the legs slightly crossed. If now the child begins to cry, and convulsive movements of the extremities are added to the unusually intensive grimaces of the face during the crying spell, the spastic condition of the extremities is seen very clearly. While the great toes were turned upwards and abducted even at rest, now all toes are spread asunder, fan-like, with great force, and the adductors of the thighs are drawn more tensely. Passive movements meet with considerable resistance, the deep and the superficial reflexes are seen to be increased. In the movements of the arms, ataxic phenomena appear. The child likes to be fondled; it recognizes the nurse, too, and may even shout joyfully upon seeing her. It seems upon close investigation to be far less mentally impaired than one should expect. The visual power, it is true, is considerably impaired. Upon ophthalmoscopic examination an incipient atrophy of the optic nerve will be found. The little patient takes the bottle willingly and quickly, the digestion, too, is good, but the greater part of the nourishment is vomited daily once or twice without, however, considerably impairing its well-being.

Symptomatology and course.—The before-mentioned disease picture corresponds to the average type of the disease. But many details need to be added in individual cases.

The disease may begin at any age, but by far the greater number of cases are observed in the first two years of life. There can be no doubt, that the great majority of these cases have to be regarded as congenital, or as developing upon a basis of congenital anomaly. A strict separation, however, can not be made here. Even in cases developing in the later years of life, it is sometimes not improbable, that congenital conditions play a rôle in their origin.

Only one class of hydrocephalic cases can be discussed separately; namely, that in which the disease has to be interpreted as the final stage of an acute meningitis. These cases represent with absolute certainty the type that we wish to designate by the name *hydrocephalus acquisitus.* The meningitic condition begins in these cases in the typical manner: with fever, spasms, stupor, nuchal rigidity, etc.; lumbar puncture demonstrates the existence of an inflammatory exudate in the meninges, rich in albumen and in purulent matter. Pus-producing micro-organisms (meningococci) may also be found. As to all the further details, we refer to the chapters on serous and epidemic cerebro-spinal meningitis. The meningitis recedes and now appear gradually, and in steadily increasing measure, the symptoms of the augmentation of the fluid in the cranial cavity. In the initial stage the higher percentage of albumen in the cerebral fluid points still to the inflammatory origin of the disease; later on, from a symptomatic standpoint, further distinction is impossible. Notwithstanding this, it is of importance to make sure in each individual case, whether it does belong to this ætiologically well-characterized group, as the chances of an improvement of the disease through operative means in these cases are slightly more favorable than in the so-called *hydrocephalus congenitus.*

The latter may be present in a considerably developed degree even at the birth of the child. The enormous cranium in such cases is a considerable hindrance to parturition, and under certain circumstances necessitates craniotomy during delivery. Anatomic findings in such truly congenital cases of hydrocephalus point to the possibility, that part of these may have their origin in inflammatory processes of the meninges, and therefore are to be regarded pathogenetically as belonging to the same class as the above-mentioned cases of acquired hydrocephalus. The beginning of the disease in most congenital cases is to be placed in the second half of fetal life; but some isolated observations make a still earlier origin of the disease likely (third fetal month, according to *Zappert and Hitschmann*).

The development of the disease in children, who do not die during delivery or shortly afterwards, varies. The head may be very striking right from the beginning, because of its volume, and this continues to increase by successive stages; or the child may at first appear entirely normal; but the growth of the cranium can be observed even during the first weeks of life; or it may be that the first months pass seemingly without any disturbance, but then insidiously an impairment of the general well-being takes place, which in the beginning can not be explained on any definite basis, only to be followed slowly in rather regular progression by the increase of the skull cap with all its consequent phenomena.

The growth of the cranium progresses very quickly at times, and by measuring its circumference at the temples it is possible to find a regular weekly increase of 1 cm. and even more; but usually the increase is much slower, especially in the cases of the somewhat older nurslings and children in the second year of life, in which the sutures are already closed. In these the closure of the fontanelles does not take place (the fontanelle may even remain open until the third decade of life). But it may also happen that the sutures are opened anew and especially the appearance of the lateral fontanelles in the more severe forms of the disease is almost the rule.

In case of a pronounced increase in the frontal part of the cranium, the great fontanelle may be drawn backwards and occupy the parietal level.

Regular comparative measurements only make it possible to judge correctly the increasing growth of the cranium and its standstill, which may occur at any stage of the disease, and usually signifies its final stage. To do this, one measures with a tape-line the temporal circumference and the space from the root of the nose over the parietal bone to the protuberance of the occiput, as well as the relation between the height of the forehead (glabella hair border) and the length of the face (glabella chin). In taking the circular measurement, the maximum transverse diameter is of special interest (biparietal diameter). In older children especially, the comparison between the circumference of the head and the length of the body is of importance. According to *Raudnitz*, the quotient between the length of the body and the circumference of the head amounts in normal children:

Newborn	1.4
One year old	1.6
Two years old	1.7
Three years old	1.8
Four years old	2.0
Five years old	2.1
Six years old	2.2
Seven years old	2.3

The circumference of the cranium keeps in normal children within the following limits (*Heubner*):

First month	35.4 cm.
Third month	40.9 cm.
Sixth month	42.7 cm.
Ninth month	45.3 cm.
Twelfth month	45.9 cm.
End of second year	48.0 cm.
Fourth year	50.0 cm.
Seventh year	51.0 cm.
Eleventh year	51.9 cm.

In cases of large hydrocephalus measures of 60 and 70 cm. temporal circumference in the first years of life are not uncommon. *Frank* measured in a child of 16 months the almost unbelievable circumference of 154 cm.

The *cranium*, as a rule, is globular and retains this shape in the less severe forms of the disease. According to *Sachs and Bókay* the globular form of the cranium is an indication of hydrocephalus externus, in which disease no other forms of cranium occur. With the increase of the quantity of fluid in the ventricles the above-mentioned changes in the cranium appear, the lateral projections in the parietal and temporal region, the decided arching of the frontal bone and the horizontal position of the tabular part of the occipital bone. But this latter feature is rarely to be found and so, too, the dolichocephalic shape of the cranium is rare. The shape of the cranium depends in addition partly upon the mechanical factors of the position of the patient in bed. It may happen, that because of this the occipital bone is entirely flattened or that the cranium assumes an asymmetrical form. The peculiar triangular or pear-like shape is frequently to be met with in the cases in which the disease is more highly developed.

The *hair* is mostly scant and thin, but there may be quite a profuse growth of hair, if the disease does not develop until the later nursing period. *The cranial bones* are in severe cases separated from each other by broad canals (like islands in the sea); they may become very thin, yield to pressure like parchment, are markedly softened at the edges especially and exhibit in their general measures a decided increase in comparison with their normal size. They as well as the entire contents of the cranium may become quite transparent, so that the whole cranial ball may flame up in a glowing red color, when a strong light is held behind it.

The fontanelles and sutures are quite tense and arched forward. Respiratory oscillations can not be observed and even pulsations can not always be discovered. Vascular bruits can be heard sometimes, are however without any significance.

The clearly marked veins upon the cranium are one of the most frequent concomitant symptoms of the disease.

The changes in the *eyes* belong to the characteristic sequelæ of hydrocephalus and not infrequently represent an early symptom. The convergent visual axes directed downwards, the protrusion of the bulbus as well as the phenomenon of the sclera becoming visible between the iris and the edge of the upper lid give the face a peculiar characteristic appearance. The opinion is generally held, that coarse mechanical causes, as the flattening of the osseous roof of the orbit, are at the base of this symptom complex. *Henoch* alone points to the possibility, that a paralysis of the oculomotor may play a rôle. At any rate in some severe cases I have found this phenomenon entirely lacking.

Horizontal nystagmus is rather frequent and strabismus is by no means unusual. In one case I could demonstrate a pronounced unilateral nystagmus.

The pupils are, as a rule, abnormally wide and frequently show a maximum dilatation as well as a total lack of reaction, but are rarely different in size. Neuritis optica and atrophy of the optic nerve are not at all common in cases of hydrocephalus during the first year, but may occur. This serious complication is the more frequent in later years.

The other cerebral nerves usually do not present any abnormal changes. But, as my experience shows, periods of regular vomiting of cerebral nature are rather frequent. Disturbances in sucking and swallowing occur but rarely. On the other hand, in serious cases, *Oppenheim's* eating reflex is present (*Fürnrohr*). Quite frequently I have observed rigidity of the nape of the neck and opisthotonos and sometimes a peculiar disturbance in breathing, loud snoring or gargling during inspiration, which seemed to me to be brought about mechanically by the opisthotonos or by changes in the base of the skull. Hearing may be intact even in the most severe forms of the disease.

As soon as the hydrocephalus is greatly increased in size, the children can not raise their head any more or keep it in an upright position; this is one of the most remarkable symptoms of the disease. With the change in the center of gravitation the head falls without resistance sidewise or backwards, as if it were a lifeless part of the body. The ability of rotating the head, when the patient is in a horizontal position in bed, is retained longer.

The rest of the body, the trunk and the extremities, is mostly behind in development and atrophic, but cases with extraordinarily strong and well-developed extremities are by no means rare, especially in children that are nursed at the breast.

Spastic *conditions* and increase of reflexes in the lower extremities are almost the rule; furthermore, I should feel inclined to regard the increase of the patellar reflex as an early symptom of the disease. As in *Little's* disease the spasms in the adductors and the inclination to cross the legs are specially pronounced. In one case I could observe, as the result of the persistent hypertonia, hypertrophy of the musculature of the calves (goblin-calves: gnomenwaden).

When the disease reaches a higher degree, the arms and the trunk become also rigid; the spasms are especially conspicuous when the children cry. At this time the toes are spread apart, the hands clenched into a fist with the thumb turned in. Tremor and ataxic phenomena may supervene. I have never observed athetosis.

During rest I have often seen the little patients stretch their arm vertically upwards with closed fist and remain in this stereotyped position, like catatonics, several minutes practically motionless.

Paralyses, even hemiplegic forms of paralysis, have been described (more frequently in older children); however, they have to be regarded as exceptional. *Convulsive attacks* of all kinds, on the contrary, belong to the most frequent complications. But in even the most severe degrees of the disease eclamptic attacks may never occur. Some of the patients cry very much and with an unnatural vehemence, probably because of severe *headache.*

The *sensibility* is mostly slightly diminished, so far as examination is possible at this tender age, but I have observed also a well defined hyperæsthesia.

The *vegetative functions* remain undisturbed with the exception of the above-mentioned vomiting and a frequent, pronounced tendency to constipation. Dentition is mostly retarded. The disease itself does not give rise to *fever*. But in cases of a large hydrocephalus a decubital phlegmon of the occiput is easily developed, which then may become the cause of death.

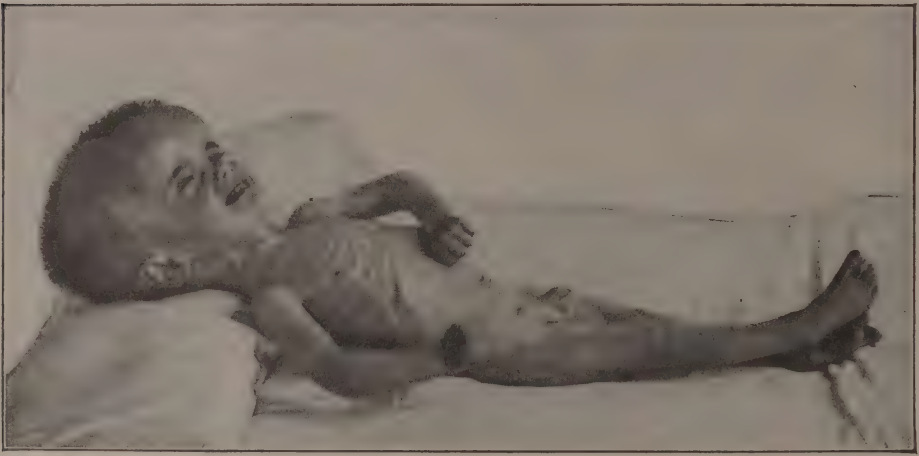

FIG. 195.—Congestive hydrocephalus in cerebral tumor (lymphangio-sarcoma). 20 months old. Symptoms of general rigidity. Bilateral atrophy of the optic nerve. (Author's observation in the Gisela children's hospital, Munich.)

In the less severe cases, that become stationary, the *psychical functions* remain not only intact, but may even develop remarkably well. Even in severe cases, one is frequently astonished to observe the considerable preservation of mental functions notwithstanding the fact that the substance of the brain is extensively destroyed; but the later consequences are usually *imbecility or idiocy*, even when the growth of the cranium becomes stationary. Of 41 children affected with hydrocephalus only five were able to attend school (*Wyss*). Speech develops late and imperfectly, the faculty of walking does not develop at all, or the gait may remain at best stiff and clumsy. In several cases sexual precocity was observed (*Bourneville-Noir, Young*).

The composition of the *cerebro-spinal fluid*, which may be obtained by puncture of the vertebral canal or even of the ventricles, deserves special consideration. The fluid is, as a rule, the color of water and perfectly clear; that withdrawn by puncture of the ventricles may contain small flakes and appear slightly turbid. In greater quantities the color assumes a slightly yellowish or greenish tint.

The reaction is alkaline, the specific gravity varies between 1001 and 1009. Formative elements are found only in very small numbers. The percentage of albumen (albumin and globulin) is very slight, 0.25 to utmost 1 per mille. If the punctures are made in rapid succession the percentage of albumen may increase considerably (*Bókay*, *Finkelstein*, my own observation). The relatively high percentage of potassium, which *C. Schmidt* has found (potassium: sodium = 1:3,18) has not been found by other investigators (*Fr. Müller*, *Salkowsky*, *Langstein*) and is explained by *Salkowsky* as a febrile phenomenon. Carbonic acid and phosphoric acid are found regularly, besides often a reducing substance probably grape-sugar. *Langstein* presumes, also galactose. Ferments also are present, diastatic ferment (*Gröber*), glycolytic ferment (*Nawratzki*).

The pressure, under which the fluid is withdrawn by lumbar puncture, may be very considerable, mostly more than 20 Hg, to 60 Hg (*Pfaundler*), but, as a rule, it is only so high at the acme of the disease and under all circumstances only when the communication between the ventricles and the vertebral canal is preserved. Otherwise only quite insignificant quantities of the fluid run off and the tension of the fontanelles remains unchanged.

The fluid withdrawn by puncture is usually replaced within a very short time; 250 ccm. and even more may collect again within a few days, and the cranial bones, which, immediately after the puncture fall together like potsherds, are again stretched tensely.

Outcome.—Death may occur in coma or amidst convulsions; but this is rather rare. Far rarer still is the bursting of the cranium. Still such cases have been described by *Veninger* and others. Usually it is complications that carry away the children (for instance, decubital phlegmons), disturbances in nutrition, or intercurrent diseases; in not a few cases, operations that have been performed to relieve the desperate condition of the little patients.

The disease in every stage may become *stationary*. In some cases, such a standstill may be regarded as a cure, though the peculiar shape of the head remains for the rest of life.

In other cases, the above-mentioned impairments of body and intellect remain. But it happens also, that months and even years after such a standstill a new eruption of the exudate takes place.

Spontaneous cures have been observed in some exceptional cases, in which the hydrocephalus through coarse mechanic traumata or the wasting of the cranial bones by pressure was evacuated externally. In such a case the fluid may drip for many days through the nose, the orbital cavity, the mouth, the ears, the frontal bone, or it may happen that larger quantities flow off repeatedly, for instance through the nose (hydrorrhœa nasalis). But such spontaneous cures are extremely rare.

Chronic Hydrocephalus of Older Children.—As soon as the sutures and the fontanelles are definitely closed, the abnormal growth of the cranium meets with energetic resistance. Therefore the clinical picture of hydrocephalus in later childhood shows quite a different appearance from that in the first two years of life; it approaches that of hydrocephalus in adults. The effect of the pressure of the fluid is far more injurious, since the skull cap can no longer yield; it is true, that it happens even in these later years, that the sutures are again reopened (*Henoch*), but as a rule the greater yielding power of the child's cranium as compared with that of the adult is exhibited only in that the cranium in toto grows faster and assumes larger proportions. Therefore in judging these conditions in later childhood the circumference of the cranium is especially to be considered.

The **symptoms** here, as a rule, are introduced by disturbances in sight and gait. Atrophy of the optic nerve appears much more frequently and much earlier than in hydrocephalus of nurslings; it may be ushered in with bitemporal hemianopsia and frequently leads rapidly to blindness. More or less severe spasms in the legs supervene, with exaggeration of the patellar and Achilles tendon reflexes. The typical picture of *Little's* disease may ultimately appear (*Ganghofner*). But there are also cases without increase of reflexes (*Finkelnburg*). Frequently extremely violent headaches occur, which may last several days and then disappear; vertigo and vomiting, tinnitus aurium and paralyses of cerebral nerves supplement the clinical picture. Rarer phenomena are sensitiveness of the cranium to percussion, cerebellar ataxia (*Finkelnburg*) and tremor (*Oppenheim*). Finally a state of stupor and loss of memory appear; in brief, the whole course of the disease may resemble in a very high degree that of a cerebral tumor, from which it may be eventually distinguished only by the rapid growth of the cranium and the occasional long lasting remissions. The determination by lumbar puncture of an increased pressure in the cerebro-spinal fluid does not suffice for making the differential diagnosis. These brief remarks must suffice here; and, as to further details, I refer to the chapter on the hydrocephalus of adults.

Ætiology and Pathogenesis.—So far, only the genesis of hydrocephalus e vacuo and of *obstruction hydrocephalus* is clear. The latter occurs when the bloodflow from the brain, especially from the vena magna Galeni is obstructed, for instance because of tumors in the posterior cranial fossa. Compression of the aquæductus Sylvii, too, may disturb the communication between the ventricles and the subarachnoid space, and lead to hydrocephalus.

But what is the essential nature of the other forms? Is it an increase in the secretion of the fluid? Is it a greater difficulty in its flowing off, are the choroidal vessels or the ependyma of the ventricles the real seat of the disease? All these matters are as yet very obscure. We know that in some cases the obstruction of the communicating passages between the ventricles and the subarachnoid space, the occlusion of the foramen Magendii or of the foramina Monroi are connected with the disease, but in many cases none of these conditions are present and the discussion is quite fruitless, because facts are lacking to serve as a foundation for it.

But one thing may be regarded as proved, namely that many cases of congenital hydrocephalus as well as of those first developing in later life are connected with *syphilis*, perhaps may even be regarded as parasyphilitic phenomena, and sometimes yield to antisyphilitic treatment.

Furthermore, it is known that in some families hydrocephalus repeatedly appears. Possibly parental alcoholism has a certain ætiological importance.

All the other statements concerning causative factors (somatic and psychic traumatism or infectious diseases during pregnancy, consanguinity, etc.) are not convincing in the least.

That serous or epidemic *meningitis* is a principal source of acquired hydrocephalus we have already mentioned.

Diagnosis.—Just as easy as the diagnosis of chronic hydrocephalus in well-defined cases is in the first and second years of life, just so difficult the diagnosis may become in later periods, and confusions with cerebral diplegias and especially with cerebral tumor may possibly be unavoidable. In the latter, however, a secondary obstructive hydrocephalus may dominate the disease picture even in nurslings.

But considerable difficulty may arise even in infantile hydrocephalus, when it concerns an early diagnosis in babies at the breast. Only regular and painstaking measurements of the cranium will prove whether the suspicion of a hydrocephalus is justified or must be discarded. To the early symptoms belong, as a rule, the increase of the patellar reflexes, and frequently also the change in the direction of vision. If to these two phenomena a conspicuous globular shape of the cranium with gaping sutures and fontanelles is added, the suspicion of a hydrocephalus seems justified. But concerning this, I wish to mention that according to my experience, in cases of premature birth the tiny infants may show for weeks and even months the typical misdirection of vision and the round shape of the cranium without the suspicion of hydrocephalus becoming justified later on. Twice only have I seen in such cases a hydrocephalus of moderate proportions actually develop; in the majority of cases, the suspicious symptoms disappeared gradually. One must therefore be especially cautious in diagnosing hydrocephalus in cases of infants prematurely born.

The *rachitic cranium* may be mistaken for a hydrocephalic one. But it is rather quadrangular, and the fontanelles do not bulge out tensely. Besides the absolute volume of the cranium is not increased very considerably, and the other nervous symptoms are lacking. That rachitis and hydrocephalus may be observed in one and the same individual is not surprising, considering the frequency of the former disease. Many authors assume that a slight hydrocephalus may develop upon a foundation of rachitis. *Stöltzner* thinks that in such cases, periosteal proliferations in the vicinity of the foramina of the base of the skull may give rise to an obstruction in the lymphatics.

Syphilitic thickenings of cranial bones will hardly ever lead to confusion, when attention is paid to the other symptoms and the hardness of the bones in the latter disease is also taken into consideration.

More likely is a confusion with the so-called turret-shaped skull (*Turmschädel*), that peculiar form of the cranium which is dependent upon disturbances in the growth of the bones at the base of the skull, and is frequently combined with visual disturbances and atrophy of the optic nerve.

The rare cases of *hypertrophy of the brain* need be mentioned only briefly. The increase of the cranial volume in these is said to affect chiefly the occiput; besides, the position of the eyes so characteristic of hydrocephalus is said to be lacking. If the fluid flows off freely, in lumbar puncture, it speaks against the diagnosis of cerebral hypertrophy.

A diagnosis of *hydrocephalus externus* is hardly possible; according to *Sachs* the protrusion of the frontal and occipital bones excludes this disease.

Lumbar or cerebral puncture makes the distinction of hydrocephalus from *pachymeningitis hemorrhagica* possible; in the latter a yellowish or hemorrhagic exudate, rich in albumen, flows off. Furthermore, in the latter disease retinal hemorrhages are found (*Göppert*) as a rule.

In *meningitis* the percentage of albumen in the cerebro-spinal fluid is by far greater, but it must be remembered that in hydrocephalus, too, repeated punctures may frequently lead to an increase in the percentage of albumen in the fluid.

The distinction between congenital and acquired hydrocephalus is frequently impossible. If other malformations exist, it speaks for the former—an acute onset with meningeal symptoms for the latter disease.

Slight pressure of the spinal fluid and early cessation in the escape of fluid combined with the other signs of hydrocephalus point to the presence of a primary or secondary occlusion of the ventricles.

Prognosis should never be made with definiteness, but is usually unfavorable, especially when the growth of the cranium is rapid. Further details may be gathered from what was said about the course of the disease. The acquired forms are more susceptible to treatment than the congenital ones.

Treatment.—**Internal treatment** with derivatives, diaphoretics, etc., according to the reported results, seem to be of no value. On the other hand, there seems no vestige of doubt that a thoroughly carried out course of *antisyphilitic treatment* (with or without synchronous lumbar puncture) has, at times, led to cures. Of course not much can be expected of small doses. *H. Neumann*, for example, had pronounced success with 0.25 iodide of potassium daily; in all he gave within nine months 75 g; in addition, at the end of the first year, treatment by inunction with mercurial ointment of one g daily; altogether 42 g was employed. Injections of sublimate were also successful (*Russo*).

One will hardly venture to apply derivative methods (seton, ointment of tartar emetic, after *Quincke's* example) to children.

I have tried several times to influence the general condition of the patient by giving adrenal preparations, but without any favorable result.

Somma has reported favorable effects from the application of rays of sunlight to the occiput, 15–20 minutes daily. Other authors have apparently not tried this treatment.

On the other hand, *surgical methods* at present dominate the treatment of hydrocephalus, and justly so, though it must be granted, that no great success has as yet been attained. It is readily understood, that even from the best methods no more can be expected, than an arrest of the disease process. The cerebral function, that has been lost, can not be replaced. Nevertheless, pure symptoms of pressure may retrogress entirely, and therefore in suitable cases, lost vision may be recovered.

Of all the procedures, the most harmless is *lumbar puncture*. It alone may suffice to produce a cure, and postmeningitic hydrocephali have been brought to a standstill in quite a number of cases through its systematic use. It is advisable to make the puncture every 3–4 weeks, and to withdraw each time small quantities, 50–60 ccm. (*Bókay*) or even 20–30 ccm. only (*Kröpfelmacher*). In many cases the puncture will have to be repeated very frequently (in the case of *Kröpfelmacher*; for instance, 66 times) and possibly combined with antisyphilitic treatment (*Slávik*).

Ventricular puncture is to be used in cases in which lumbar puncture does not lead to an escape of greater quantities of the fluid. In this way considerable quantities can be withdrawn, but usually a rapid restitution of the former condition will soon be witnessed.

In children with open fontanelles the operation presents no difficulties. Symptoms endangering life (collapse, convulsions) may occur if the outflow of the cerebral fluid takes place too rapidly, and in such a case, it may possibly become necessary to inject physiological salt solution and stop the operation. One will hardly feel justified in injecting afterwards diluted tincture of iodine (10 g Tct. iod. 20 g water).

Quite a number of surgical methods have been devised to insure *continuous drainage of the ventricles*, but we can not give the details here; free external drainage mostly carries the child off quickly. Drainage under the galea (*v. Mikulicz*) has not as yet been tried very often. It has recently found several sponsors. The experiments made by *Payr*, to effect the drainage by means of freely transplanted blood vessels appear most promising. It is said, that in this way even a direct connection between the lateral ventricles and the longitudinal sinus may be brought about. The formation of a connection between the ventricles and the sub-dural space by means of catgut threads, or glass wool, is said to have been of advantage occasionally. *Van der Hoeven* recently reported satisfactory functional results in two cases concerning older children, in which he scarified the dura after trepanation of the occiput.

Compression of the cranium by strips of adhesive plaster or other similar contrivances only serve to increase the injurious symptoms of pressure.

To summarize, therefore, we may say, that in the acquired forms of hydrocephalus repeated lumbar punctures are to be recommended; they should be replaced by ventricular punctures only in case of a disturbance in the communication with the cerebral cavities; that in the so-called congenital forms the same kind of treatment is to be applied as soon as possible; and that, if no success is observed, continuous drainage ought to be attempted. Operative interference is not to be recommended (*Finkelstein*) in the more advanced stages (idiocy, etc.) of either stationary hydrocephalus, or congenital hydrocephalus. In the latter case, at most, as a symptomatic expedient.

In the care of nurslings a prevention of decubitus must be especially considered; in cases that take a favorable course the motility of the extremities has to be improved by massage and warm baths. These children naturally, later on, need special mental supervision, to develop what has been left of their intellectual faculties.

Spina Bifida (Rachischisis)

By Spina bifida we mean a fissure formation in the vertebral canal which may be combined in different ways with similar fissures in the outer integuments, the spinal membranes and even in the spinal cord itself. In the slighter cases the unbroken outer skin covers the vertebral canal, which is not perfectly closed (*spina bifida occulta*). The affected part may be noticeable because of a flat cushion-like protrusion. It frequently shows a zone of abnormal hair growth. The seat of this form of fissure formation is usually the lumbar region.

In the majority of cases, however, there appear hernia-like protrusions of the contents of the vertebral canal, in the shape of globular or ovular broad-based or pedicled tumors, and varying in size (from the size of a nut to that of a child's head). As a rule they are located precisely in the median line of the back. The seat of the tumor is, as a rule, the lumbar, or sacral region. But tumors, located higher, also occur. In the back of the neck, especially, they are not rare (*spina bifida cervicalis*). The tumor is invariably filled with cerebro-spinal fluid, and in addition contains varying parts of the spinal cord and of its membranes.

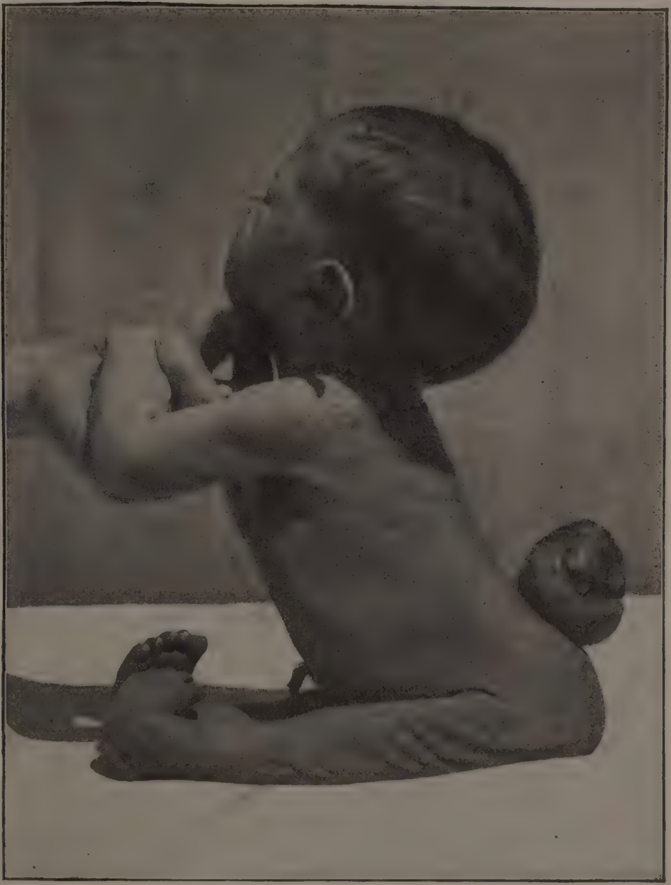

FIG. 196.—Spina bifida sacrolumbalis. Meningomyelocele. Clear demarcation of the zona dermatica. Club feet; imperfect development of the musculature in the lower extremities. Paralysis of the sphincters. (Author's observation at the children's clinic at Heidelberg.)

This malformation, which has been studied carefully especially by *v. Recklinghausen*. is in its embryological aspect to be explained by the fact, that the union of the medullary line, which usually takes place at an early stage of embryological development, remains incomplete at some point.

In the rarest form, the *meningocele*, the tumor does not contain any portion of the spinal cord, but consists exclusively of a protrusion of the meninges. *Myelocystocele* is so formed that the central canal at the place of the fissure of the spinal column bulges out extensively, while the dorsal part of the closed spinal cord takes part in the formation of the hernia. Finally, in the most severe form of the affection, the spinal cord itself participates in the fissure formation (*myelocele* or *meningomyelocele*); it freely protrudes outwardly and constitutes the uppermost part of the tumor. In these severe cases three zones can be recognized on the outer side of the tumor: first, a dark red granular-like layer, rich in vessels (*zona medullo-vasculosa*); this represents the spinal cord, and from here the somewhat wasted nerves course downwards into the vertebral canal. Adjoining this zone peripherally is a thin vesicle-like zone with a grayish tint, which from an embryological standpoint is to be interpreted as the pia spinalis (*zona epithelo-serosa*); finally, at the base, one finds the normal skin, which covers the tumor in varying extension (*zona dermatica*). As a rule, the tumor is tense, and part of its contents can often be forced back into the spinal canal by pressure. The fissure in the vertebral column may, as a rule, be detected by palpation, and always with certainty, by the X-rays. In very rare cases a spinal cord hernia may be formed through a fissure of the anterior wall of the vertebral canal (*spina bifida anterior*) and protrude into the pelvis. Frequently spina bifida is accompanied by other malformations, especially by hydrocephalus internus, but also by exstrophy of the bladder, by congenital hernias, etc. According to the investigations of *E. Schwalbe and Gredig*, anomalies in the development of the cerebellum and the medulla oblongata are frequently combined with rachischisis. In myelocele, the lower extremities, as a rule, show malformations (especially club foot).

Symptomatology.—The clinical phenomena may be very varied according to the seat and the character of the tumor. In severe cases the legs are deformed and totally paralyzed. However, the ileopsoas is usually spared in these cases. Sensibility may be entirely lacking in the paralyzed parts; serious trophic disturbances of the skin are the consequence (ulcers upon the buttocks and the genital regions). If the seat of the malformation is in the lumbar or the sacral region paralysis of the sphincter ani and also dribbling of the urine are usually present. The anus, because of the paralysis of the muscles of the pelvic floor and of its sphincter, shows a peculiar appearance. The crena ani is smoothed out, the anal region bulges out, the deep red mucous membrane of the rectum is more or less protruding. The reflexes are entirely lost; I have, however, observed the peculiar phenomenon that touching the leg at any place is invariably followed by a reflex contraction of the only functionating muscle (extensor digitorum communis). If the seat of the tumor be at a high level the abdominal muscles may be totally paralyzed, in case of a lower location of the tumor the recti abdominis usually are spared. The paralysis is almost always flaccid and atrophic. The electrical reaction is mostly abolished, but may sometimes show reactions of degeneration, or indeed, be normal.

In less severe cases the paralysis may affect only the sphincters or possibly in addition the musculature of the feet.

The latter is especially true of *spina bifida occulta*, which affords considerable neurological interest. It is remarkable, that in this form of the disease the disturbances (pains, paralyses in the feet and symptoms of incontinence) appear first in a more advanced age of the children and even sometimes after the stage of puberty is reached, which may lead to serious mistakes. This late appearance of the disturbances is probably connected with traction upon the connecting strand between the spinal cord and the integument, caused by the physiological moving up of the conus terminalis in the vertebral canal during growth of man (*Katzenstein*).

Diagnosis.—Myelocele can not be mistaken, if the above mentioned peculiarities are present. The other forms are not so easily recognized. If a perfectly transparent tumor be located on a pedicle and paralyses are lacking, the diagnosis myelocele seems probable, while lack of paralyses in the presence of disturbances in sensibility and a broad protrusion of the tumor suggests myelocystocele.

The differentiation from other medially located tumors may afford some difficulty, if paralyses are absent. Teratomata and lipomata are to be considered. Combinations of lipomata and spina bifida have also been reported. I remember a case in which the diagnosis of a large abscess caused by vertebral caries could only be made by means of the diagnostic needle. If the tumor can be replaced in part a pathognomonic sign for spina bifida is present.

Of special importance is the diagnosis of spina bifida occulta. Formation of a club foot, symmetric slowly increasing paralyses of the feet, especially phenomena of increasing incontinence or of its development at a later period will rouse the suspicion that a fissure in the vertebral canal has been formed, which gains considerably in probability, if the lumbar or sacral region exhibits abnormal growth of hair. In some cases a definite diagnosis can be made only by an X-ray.

Treatment.—Treatment can only be operative. It consists in the removal of the tumor with the greatest possible care to spare the nervous elements and, when possible, an osteoplastic covering of the defect. I refer in this respect to the handbooks on surgery. If severe paralyses exist, a radical operation is contra-indicated. *In less serious cases, on the contrary, it is to be recommended by all means.* Occasionally the simple puncture of the sac has favorably influenced the paralyses (*Demme*). According to the general opinion in cases of combination with hydrocephalus an operation should not be performed. The remarkable fact has to be mentioned, that not infrequently after the operative removal of the tumor a hydrocephalus has developed. In the case of spina bifida occulta surgical help may be very successful by the removal of the strands, which drag upon, and of the fibrous plaques that compress the spinal cord.

In inoperable cases nothing can be done beyond a careful nursing of the patient; this may prove very difficult, as an infection of the moist, granulating and easily infected malformation by the feces of the child can hardly be avoided. The children die as a rule in the first weeks of their life of a purulent meningitis, an infection of the urinary tract or of the integuments or of intercurrent diseases. If they live longer the tumor may be fully covered by the integument proliferating from its edges.

Supplementary to the above we may mention here the *encephalocele*. In this disease it is a matter of quite analogous hernia-like protrusions of the cerebral substance through circumscribed cranial fissures. They are almost exclusively located at two places, at the nape of the neck or at the root of the nose, are covered by the normal skin or by a thin glossy integument and are rarely pure meningoceles. As a rule the tumor is filled with cerebral substance and usually a ventricle protrudes into the tumor (encephalocystocele). Clinical symptoms on the part of the nervous system may be lacking; idiocy, strabismus, atrophy of the optic nerve and other phenomena may be present. The treatment is surgical and consists of the removal of the tumor, the replacement of its contents and the closure of the cranial fissure.

3. INHIBITIONS OF DEVELOPMENT

We wish to treat in this chapter of several practically important conditions and forms of disease, which are to be regarded essentially as *inhibitions in development* of the infantile brain or of its parts. We omit here a number of more serious malformations, which make maintenance of life impossible as for instance, *acephalia*, *anencephalia*, *hemicephalia*, *arhinencephalia*. Such malformations are, it is true, of the greatest importance for neurological investigations and they are duly appreciated in this respect since the fundamental investigations of *v. Monakow* and his disciples have brought to light their importance. Clinically, however, they do not afford any considerable interest. *Porencephalia*, which can not be regarded as a uniform concept and does not afford a uniform characteristic clinical picture has already been treated, while discussing the pathological anatomy of infantile cerebral paralysis. We will refer to this subject again in discussing idiocy. *Hydrocephalus*, which in many cases is congenital or is to be traced back to a congenital predisposition, has been treated extensively in the former chapter, where we discussed also *encephalocele* and *spina bifida*.

We shall describe here first two clearly defined disease pictures belonging to this group of inhibitions of development, *congenital nuclear defect* (*infantile nuclear wasting*) and *amaurotic family idiocy* (*Tay-Sachs' idiocy*); finally some summarizing remarks about *imbecility and idiocy* in general will not be out of place, and they will be followed by a short discussion of *microcephaly and mongolism.*

Congenital Functional Defects of Motor Cerebral Nerves

(*Congenital Nuclear Defect, Infantile Nuclear Atrophy*)

In the region of the cerebral nerves, especially in that of the external eye muscles, paralyses occur, which are partly of congenital origin, partly develop in early infancy (only exceptionally at a later period). They are characterized by the fact, that they attack selectively, frequently symmetrically on both sides, individual nerve regions or parts of such, that no disturbances other than motor are to be observed, and that the paralysis, in cases in which it is not fully developed at the birth of the infant, progresses quite slowly to its full climax, finally that a progression into other regions of the nervous system never takes place. The disease has reached its limits and remains, as it is, without further change for decades. The electrical reaction of the paralyzed muscular regions is diminished or entirely abolished. Reaction of degeneration in the typical, congenital cases does not occur.

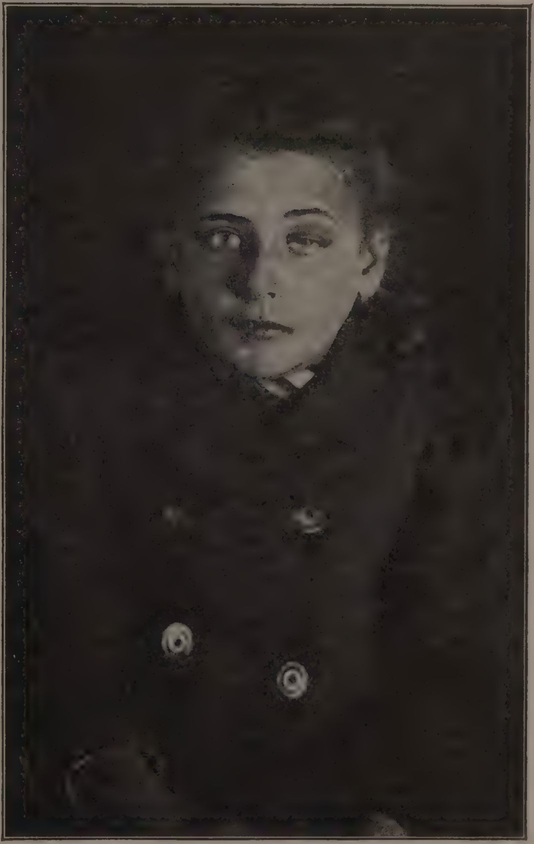

Fig. 197.—Congenital nuclear defect. Ptosis at the left; high position of the left eye-brow. (Author's observation at the Gisela children's hospital, Munich.)

Most frequently it is a matter of unilateral or bilateral ptosis. It may form the only symptom of the disease or combine with paralyses of other muscles of the eye. A complete ophthalmoplegia externa may exist and besides this a paralysis of the facial nerve, paralysis of the abducens, unilateral or bilateral, combined with that of the facial or without it may occur.

The tongue may also be involved and an interesting observation of *Heubner* points to the probability that in some rare cases the glosso-pharyngeal and the vagus, too, may exhibit analogous disease phenomena (gradually increasing deglutition paralysis).

Insufficient lachrymal secretion has also been observed in such conditions. Furthermore the frequently described phenomenon deserves mentioning that the drooping eyelid in unilateral ptosis is raised energetically as soon as the mouth is opened wide, only to droop again with its closure. It is also interesting, that in congenital paralyses of the abducens secondary strabismus may be entirely lacking (which is a usual symptom in acquired permanent paralyses). Diplopia rarely ever occurs, not even in the forms which originate after birth.

The above described disease pictures have been recognized as a group of diseases induced by one and the same cause by *Möbius;* he was of the opinion, that they were the result of nuclear wasting in the medulla oblongata. With *Kunn*, we separate the cases into two categories, congenital ones and those that are acquired in early childhood or even infancy. The former are not caused by a nuclear wasting, but by a nuclear defect, an agenesia or aplasia as the autopsy in the case of *Heubner* showed; but, as *Neurath* has stated, the nucleus may be perfectly normal. The functional loss then must be traced to primary defective development of muscles. That a congenital injury of the nerve may lead in its further course to similar symptoms is proved by the observation of *Marfan* and *Armand-Delille;* in the case described by them the paralysis of the facial was the result of a malformation of the petrous bone.

It is worth mentioning, that with these congenital forms frequently other malformations are combined, defective development of the hands and feet, congenital organic defects of the heart, especially also defects of muscles in the body, as for instance of the pectoralis major. Of course, in older persons it may be impossible to decide whether it is a matter of a congenital or of an acquired form of the disease. Besides it is hardly likely that the two types are essentially different. In the latter type it is probably in the sense of *Edinger's* "using up" theory a matter of a functional destruction of the organs, which at birth were of more or less impaired efficiency, while in the former form the organs have never functionated at all.

As to the factors of ætiological importance, we do not know anything certain. Alcoholism and syphilis (*Gierlich*) are suspected as causative factors. Hereditary and familial cases have been repeatedly observed.

From a diagnostic standpoint, it is to be remarked, that under certain conditions injuries through traumatism during birth (forceps) may produce identical clinical pictures. Bone marks may in such cases point to the ætiology. In some rare cases solitary tubercles may transitorily produce similar symptoms. Post-diphtheritic paralysis too may occasionally lead to confusion. The non-progressive character of the disease makes the differentiation from infantile progressive bulbar paralysis and progressive muscular dystrophy easy upon longer observation. In the rare cases which do not develop until childhood has passed, an eventual confusion with tabes must be guarded against. The diagnosis is important, because the prognosis is dependent upon it. The disease, it is true, is not amenable to treatment. But once the paralysis is fully developed, no further progression of the disease into other nerve centers is to be feared.

Amaurotic Family Idiocy (Tay-Sachs' Idiocy)

Amaurotic family idiocy, which was first described by *Sachs* in 1898 and has since then been observed a number of times, could be classified with the familial forms of cerebral diplegias. But it is distinguished from these by the extreme constancy of its clinical course and, so far as our present knowledge goes, also by something far more important, by uniform and typical pathologic-anatomical findings;[1] it presents therefore the best characterized disease type in the group of cerebral infantile paralyses.

Etiology.—The disease is absolutely familial, attacks as a rule several children of the same parents; in addition it is quite peculiar, in that it shows an outspoken predilection for the Jewish race. More than four-fifths of the observations were made in Jewish families, especially in families of poor Polish and Russian Jews. Neuropathic predisposition seems to be of some import.

Symptomatology.—The disease usually sets in in the second half of the first year of life, sometimes even as early as the third or fourth month. The child, which so far has been healthy and bright, becomes more quiet, languid, no longer laughs, sleeps a great deal. Slowly a decrease of visual power is observed; its eyes no longer follow any bright objects; the motility of the limbs diminishes. Examination by a physician in most cases shows already complete blindness. An examination of the fundus of the eye shows a characteristic ophthalmoscopic picture; the optic nerves exhibit a more or less developed degree of simple atrophy; *the region of the macula lutea shows a circumscribed grayish white discoloration of about double the size of the disc; in the centre at the place of the fovea centralis there is a cherry red spot.* This feature is pathognomonic for our disease. Frequently nystagmus and pupillary difference are found, occasionally also strabismus. Abnormal timorousness has been observed, sometimes also deafness.

The paralysis, which gradually affects the whole body and most severely attacks the musculature of the back of the neck, is always bilateral; it is usually flaccid, but may also become spastic. The reflexes do not show a constant behavior, they are frequently exaggerated. The mental faculties vanish more and more, until finally a complete dementia is present. The children invariably die; as a rule toward the end of the second or third year of life.

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[1] It is still doubtful, whether the case described by *Huismans* is to be regarded as belonging to our disease.

Pathological Anatomy.—In the brain macroscopically no changes at all or only very insignificant ones can be found (abnormalities in the convolutions, underweight). Microscopical examination shows a process of quite diffuse degeneration of the ganglia cells, affecting the whole gray matter of the central nervous system especially of the cerebral cortex, characterized by a peculiar swelling and bulging out of the entire body of the cell or only of some of its parts (*Schaffer*). Inflammatory changes are lacking. The retina also shows degenerative processes in the cells of the granular layer. The red spot at the fovea centralis corresponds to the normal color of the retina, as here the cells are absent.

It seems that we have to interpret this affection also as a "using up" disease, a functional degeneration of the abnormal, weakly predisposed nervous system incapable of regeneration.

Diagnosis.—If one is conversant with the disease, its recognition is not beset with difficulties. Jewish extraction, familial appearance, the decrease of the visual power arouse suspicion, the examination of the background of the eye is decisive. It is well to know, however, that in some cases, which because of their familial relation and the whole course of the disease could with certainty be diagnosed as the above disease, the macula phenomenon was lacking (*Koller*, *Heveroch*, *Mühlberger*). In two of these cases the little patients were of Christian extraction.

The *prognosis* is absolutely unfavorable. But the parents may at least be given this consolation, that the disease need not necessarily affect all their children. If a child has passed the age at which his brothers or sisters were stricken, it has a good chance of remaining spared.

Treatment.—From the standpoint of prophylaxis one might recommend that the children be nursed by a wet nurse instead of their mother.

The Juvenile Form of Amaurotic Family Idiocy

In children of a more advanced age (4th to 16th year) disease pictures sometimes are observed, which show many analogies to *Tay-Sachs'* idiocy and very likely are to be interpreted as lighter forms of the same disease (*C. Vogt*). Here, too, it is a matter of exclusively familial affections of a distinctly progressive character, which lead to blindness, diplegic paralyses and gradually to dementia. The distinction from the above mentioned infantile form is principally to be found in the absence of the ophthalmoscopic phenomenon in the macula lutea and in the slower progression of the disease. The onset of the disease in the children of the same parents usually occurs in the same year of life. Ophthalmoscopic examination shows simple atrophy of the optic nerve, sometimes even a normal disc, so that the cause of the blindness must be sought in the central optic neuron (*Vogt*). *Spielmeyer* found a retinitis pigmentosa.

That both groups belong together is the more probable, since in the cases of *Spielmeyer* the pathologic-anatomical findings showed a great similarity to the typical changes in the infantile form of the disease. Possibly connections with other diseases exist also. *Higier* found in one family two children suffering with familial optic atrophy, two with familial cerebellar ataxia and the youngest with typical *Tay-Sachs*' amaurotic idiocy. Further information will doubtless be obtained by pathologic-anatomical investigations.

Imbecility and Idiocy

By imbecility and idiocy we designate the two forms of mental weakness, that are the result of a defective development of the intellect. The less severe degrees are referred to as imbecility. A strict criterion of demarcation from the more severe forms does not exist. Such persons are frequently spoken of as imbeciles, who are still able to follow some vocation in life. The name feeble mindedness is given to the lighter degrees of imbecility.

The great group of idiocies comprises a great number of diverse conditions which have not been sufficiently investigated as yet. Many of the varieties of disturbances, which strike the cerebral cortex, may lead to impairment of the mental faculties. According to the type of these affections, different forms of idiocy are distinguished: hydrocephalic, microcephalic forms, idiocy consequent upon cerebral paralyses, upon meningitis, simple idiocies, which, for instance, may be regarded as simple inhibitions of development or hypoplasias, and besides these as special clinically well defined forms amaurotic family idiocy and myxedematous and mongoloid idiocies.

The somatic phenomena of the hydrocephalic cases as well as of those connected with cerebral paralyses have already been described. We frequently find the symptoms of the latter in idiotic children merely suggested in the form of slight spasms, increased reflexes, insignificant choreic or athetotic movements. We have ample reason for the assumption, that also in a large percentage of the idiots, in which such signs of infantile cerebral paralysis are lacking, the same morbid processes must have occurred in the brain, but that by some chance the motor regions had not been affected by them. These are the so-called cases of "cerebral paralysis without paralysis" (*Freud*). We have to deal here, therefore, with coarser malformations or destructions of the brain, with porencephalias, cystic, cicatricial or even diffuse sclerotic processes. All we have said about the ætiology of infantile cerebral paralysis is therefore valid also in these cases. On the other hand there are cases also, in which such palpable disturbances can not be found.

in which the backward condition, the aplasia of the cerebral cortex can be recognized only by a painstaking microscopical investigation. The weight of the brain is always more or less below the normal. Ætiologically such forms are frequently connected with syphilis; alcohol, also, especially procreation during drunkenness plays here a special rôle, so also neuropathic and psychopathic predisposition, which are of great importance; consanguinity of the parents and acute infectious diseases are mostly only to be added as further factors.

The shape of the cranium may vary very much; besides the relatively rare hydrocephalic types abnormally small crania are found frequently; the frontal portions especially are frequently flattened (receding forehead, aztec type); dolichocephalic types (scaphocephalic) also occur. Signs of lessened somatic capacity in other respects are frequent, especially abnormal formations of the genital organs (cryptorchidism, phimosis, hypospadia, etc.) and also abnormalities of the eyes, ears, of the growth of hair, the formation of teeth, etc.

In the cases, too, which can not be regarded as belonging to infantile cerebral paralysis, convulsions in the nursing age and later are by no means uncommon. Frequently the children exhibit quite a pronounced flaccidity of the muscles; they can not keep the head straight, can not sit and, when permitted to sit alone and thus left to the effect of gravity, may fall in any direction. A very frequent symptom is restlessness. Without pause or rest such children toss around in bed or move their limbs constantly in all directions; if they can walk, they run around restlessly in all the corners of the room from one object to another; nothing interests them, nothing stops their imperative motions (*versatile idiocy*). A clinical contrast to these patients are the *torpid or apathetic idiots*, who, untouched by the varying changes of the goings on about them will sit for hours in bed, sometimes without any motion whatsoever, and stare around vacantly giving vent to animal cries from time to time or if they are disturbed in their comfort, giving forth a loud howl.

Most idiotic infants, even those who are severely affected, recognize at least their mother or nurse, are capable of tender emotions and appreciate being fondled. Their uncleanliness is an especially great nuisance; some of them never learn to voluntarily control the discharge of urine and feces, but besides this, one sometimes may note, how they really seem to delight in spreading their excrements over the bed; even cases of coprophagia have been reported.

A characteristic of most low grade idiots is their constant slavering. It is frequently artificially furthered in that the children move their little hands to and fro in the mouth, the hand sometimes disappearing to the wrist in the buccal cavity; even in nurslings can this peculiarity be observed. Many idiots must be fed all their lives. The sensibility is, as a rule, considerably dulled. Pricking with a needle does not bring forth any reaction to pain.

Sitting, standing and walking is mostly acquired in later years; in the cases of general diplegia, however, walking is rarely learned, if idiocy exists. The faculty of speech may be present in a rudimentary form and even develop considerably, notwithstanding the serious impairment of the higher mental functions.

All these disturbances naturally may be present also in lighter and the lightest degrees.

Masturbation is common in idiots. We can not enter more in detail into the disturbances of mental activity within the scope of this book. It is well known that memory itself may be especially well developed in imbeciles along some lines and that the defective faculty of judgment may be accompanied by unpleasant characteristics, such as arrogance, inconsiderateness, brutal behavior, etc. Many imbeciles become criminals.

A special form of idiocy is that dependent upon *psychic deafness*. According to *Heubner* these cases are characterized by the physical development being less impaired. The children learn to sit, to walk, etc., at the proper period, the sensations are present as well as the elementary auditory impressions; even echolalia has been observed. But the acoustic images of words are lacking. The patients are unable to connect a conception with the word and since the acquisition of elementary as well as abstract conceptions are commonly dependent upon hearing, no mental life can be developed in them. Mostly it is the versatile idiots, that belong to this category. Prognosis is said to be less unfavorable in these than in the other cases "if the children are brought up by the proper kind of education, which first tries to induce them to collect conceptions in other ways, than the usual one by means of hearing."

Diagnosis.—Diagnosis of the individual ætiological forms of idiocy, as far as it is possible, is tantamount to the recognition of the cerebral paralyses, the hydrocephalus, the turret shaped skull, etc. Concerning tuberous sclerosis see page 761. The practically far more important question concerning the degree of mental weakness, whether idiocy, imbecility or feeble-mindedness can only be solved by studying the child more minutely and usually in later years only. As to methods and numerous details we refer to the excellent booklet of *Ziehen* "Die Geisteskrankheiten des Kindesalters." The recognition of idiocy in the nursling or first infantile period deserves special consideration. An important sign is here the defective or lost reaction to needle pricks (*Thiemich*); it may strike one as peculiar furthermore, that the child does not learn to "fix" an object, that its eyes do not follow brilliant objects as the eyes of healthy children do, that it does not try to grasp the objects, that it does not greet the mother or the nurse with a friendly look or a smile, does not recognize them by their voice, that taken all together it does not know how to get into relation with the objects and people surrounding it. In the lighter cases the lack of speech development often is an indication; but it must be remembered, that not infrequently children, that are perfectly normal mentally, do not commence to speak until the second and third year and even still later. Of much greater importance is the proof, that the child understands the meaning of the spoken word and connects conceptions with it. Of special importance is the recognition of pure deaf mutism as such. Many of these unfortunate creatures, who, if their affliction is rightly recognized, are certainly capable of being instructed by proper educational methods, are wrongly regarded as idiots and therefore neglected. Mental debility frequently can be recognized only at the time of attending school. Long persisting enuresis, masturbation may represent symptoms of such and arouse suspicion. Moral deficiencies, as a rule, are to be traced back to lowered mentality only, if physical or intellectual defects can be proved present, furthermore, if having been led astray, neglect and other factors may be excluded as causative agencies and if neither punishments nor rewards show any effect upon the child (*Ziehen*).

Prognosis.—It is always doubtful, though the modern educational success of special pedagogy, which has first been systematically carried out in France (*Séguin, Bourneville and others*), has made the prognosis much more favorable than before. These methods have helped many children with undoubtedly lessened mental capacity to complete mental maturity and have made even imbeciles into at least somewhat useful members of human society. As things stand today, however, a favorable pecuniary situation of the parents is an essential condition of profiting by these modern educational methods.

Therapy.—The treatment can be causal only in the hereditary syphilitic forms. As to the rest, the children with mental deficiency must be preserved from any damaging influence—to which frequently the neuropathic milieu of the parental home may belong—from alcohol, bad society, etc. If the mental weakness reaches a somewhat higher degree the child ought to be sent to special educational institutions, but one should emphasize the necessity of doing it at an early period, as the results are the most favorable, when expert treatment begins in the third or fourth year of life. We cannot enter here into the details of the pedagogic methods useful in such cases.

Microcephaly

By microcephaly we designate an abnormally small size of the cranium and its contents. In adults the usual maximum is a cranial circumference of 45 cm. and a brain weight of 900 gr. In children there is no fixed standard; one must judge by comparison with the measurements of healthy children of the same age, whether the cranial measurement remains considerably behind and also by the characteristic shape of the skull.

Microcephaly is a consequence of an inhibition or disturbance in the normal growth of the brain and the disease is congenital in by far the greatest number of cases. The cranium adapts itself in size to its contents. The assumption that a premature ossification of the cranial sutures is the primary cause of the disease has long since been refuted. The inhibition in the development of the brain can only be strictly endogenous or primary, an arrest at primitive stages of development, in which under certain conditions the cerebral formation may resemble that in apes or carniverous animals; as a rule it is accompanied by an abnormal distribution of the gray matter (heterotopia, metaplasia) and sometimes by abnormal types of convolutions (macrogyria, microgyria). These simple inhibitions of development are designated, following *Giacomini*, as *microcephalia vera*. Contrasted to these are the *pseudo-microcephalies;* in these cases it is a matter of pathological processes, for instance inflammation, which affect the brain at a later period of its development and causes severe changes, that as a rule, are already macroscopically visible. These disturbances however may occur at such an early period of fœtal development, that they can only be discovered by a most painstaking microscopic investigation. Because of this the concept of microcephalia vera, which had only a relation to the macroscopic appearance of the brain, is considerably restricted. In the genuine as well as the non-genuine microcephalies the spinal cord, too, is considerably diminished in thickness (agenesia or hypoplasia of the pyramidal tracts).

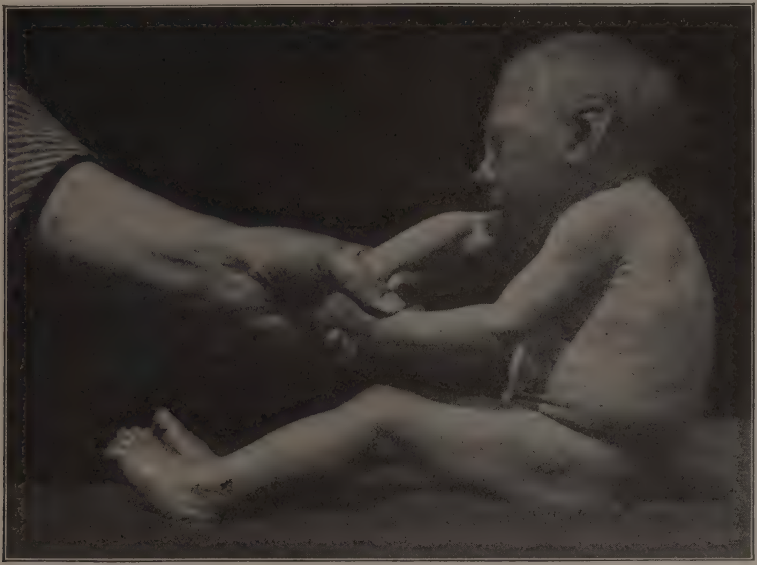

FIG. 198.—Microcephaly with general rigidity. 5 months old. Temporal circumference 33.3 cm. Congenital atrophy of both lenticular systems. Vertical nystagmus from time to time. (Author's observation at the children's clinic Heidelberg.)

The facial type of the microcephalic is, as a rule, very characteristic. The little head and the receding forehead combined with the frequently very strongly developed nose remind one of a bird's face. The intellect, as a rule, is very poorly developed, but the most hopeless idiocy is usually found in pseudo-microcephaly, which almost invariably, in the more severe forms, must be classified with the congenital cerebral diplegias. Usually the children exhibit the symptom of general rigidity in a very pronounced degree. On the other hand, however, there are cases of microcephaly, where the afflicted are quite active and mobile; this form appears mostly familially and corresponds more than the others to microcephalia vera. In those cases in which pronounced spasms and especially athetosis are found, it is almost always a matter of pseudo-microcephaly (*Ibrahim*).

The ætiology of the latter disease as well as its clinical phenomena are the same as those of prenatal cerebral paralyses. It is noteworthy, that the children with the tiny heads not infrequently are born after difficult parturition. Frequently one observes several months after birth prominent ridges and elevations at the place of the sutures, which however do not necessarily permit the conclusion, that an absolute ossification has taken place. The great fontanelle, from the beginning abnormally small, closes very early, after six months or probably even at the end of the third.

This condition can not be influenced by treatment. The operative treatment inaugurated by *Lannelongue* (craniectomy or craniamphitomy), which was said to further the growth of the brain by preventing the supposedly premature ossification of the skull cap, was based upon wrong premises and did not show any real successes.

Mongolism

The term mongolism denotes a peculiar type of idiocy showing some similarities to myxedema and sporadic cretinism, though as yet no definite connection with the thyroid gland has been found, as this organ has been proved to be perfectly normal macroscopically and microscopically in children afflicted with the disease. The name of the disease is due to the peculiar facial type; the face is broad with slightly protruding cheek-bones, the nose is flat and stubby, the root of the nose sometimes sunk in, saddle like, the eyes are set obliquely and have a somewhat slit-like appearance; the mouth is usually kept open and permits the frequently thickened, fissured tongue to hang out. The skin is fat and soft; the joints and muscles are abnormally flaccid, hands and feet plump and not rarely slightly malformed. Constipation and umbilical hernia are frequent. Speech is mostly undeveloped and hoarse; the growth of the body is normal, the closing of the fontanelles usually retarded, the cranium mostly small and brachycephalic. Congenital cardiac defects are strikingly frequent. The intelligence is but very slightly developed; the children do not learn to walk till late, speak little and soil themselves for a long time. A certain degree of versatile unrest is the rule with them.

As yet only few cerebral findings are known. According to *Vogt* the disturbance is due to an inhibition of the last stages of the embryonic cerebral development.

It may be difficult to make a sure diagnosis. Differentiation of the disease from myxedema and cretinism is as a rule easy. It is based upon the agility, the absence of dwarfism and of myxedematous cutaneous changes; in doubtful cases an X ray picture is decisive; it shows at best only a slight retardation in the appearance of the bone nuclei, in sharp contradistinction to the high grade backwardness of ossification in cases with thyroid defect.

The condition of the patients, as has been empirically proved, can be influenced favorably through medication with thyroid preparations, but to a far less degree than in myxidiocy. The favorable effect is especially observed at the beginning of the treatment and slackens afterwards; there are also cases which cannot be influenced at all and have to be treated according to the general rules governing the treatment of idiocy.

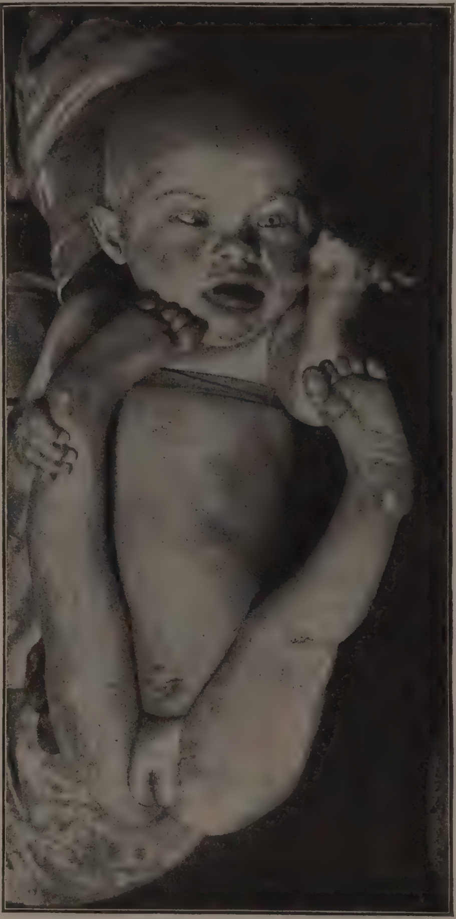

FIG. 199.—Mongoloid. (*From Pfaundler*)

4. POLIOMYELITIS ANTERIOR ACUTA (ACUTE SPINAL INFANTILE PARALYSIS)

Poliomyelitis anterior acuta is an affection of the spinal cord, especially of its anterior horns, mostly restricted to childhood; it has an acute onset and a non-progressive course and leaves, as a rule, permanent flaccid paralyses in part of the affected groups of muscles.

Ætiology and distribution.—Infantile spinal paralysis is quite a frequent affection of childhood. As a rule it appears sporadically, the special ætiology of which is mostly quite obscure. But more and more frequently in recent years smaller or larger epidemics have been described in quite different countries (France, Sweden, Norway, Finland, Germany, Austria, Switzerland, Italy, America, etc.).

The northern countries especially have been visited frequently by severe epidemics, one of which (1905) furnished more than 1000 individual cases (*Wickmann*). A still larger epidemic with more than 2000 separate cases occurred in 1907 in New York and its vicinity. It seems that the form of the disease which occurs in epidemics is to be regarded in all respects as identical with that which occurs sporadically. But, as a rule, the distinctive feature of the epidemics is that adults and children of more advanced age are attacked more frequently than when the disease appears sporadically. Probably this is due to an increased virulence of the excitant of the infection, a point indicated also by the increased mortality. But on the other hand, during the observation of such epidemics, abortive forms of the disease have been reported, as well as occasional combinations with other nervous diseases (polyneuritis, encephalitis, Landry's paralysis); to the latter *Medin* was the first to call attention; all this should preclude us from a too schematic interpretation of the real nature of the disease.

That we have to deal with an *infectious disease* can hardly be doubted, after what we have already said. This was pointed out before with regard to the initial stage of the clinical picture (*Strümpell*), the acute febrile onset and the marked impairment of the general state of health, as well as by the statement made first by *Sinkler*, and often confirmed since, that a cumulative appearance of poliomyelitis always occurs in the summer months (July and August).

We ought to mention here also some experimental experiences in this respect, the detailed explanation of which would lead us too far, especially as no one has succeeded as yet in producing experimentally a clinical picture and changes which could be regarded as identical with those of poliomyelitis (*Wickmann*).*

As to the *excitant of the infection* and its place of entry into the body, we know as yet no more than about its manner of working. No one has succeeded thus far, not even in the most recent cases, to discover the microorganisms in the affected foci of the spinal cord through either a staining or cultural process; this may be due to the fact that the microorganisms in the spinal cord can not be discovered by our present methods of investigation, even quite soon after death, as numerous experimental tests have proved (*Homèn*). But on the other hand, one must also consider the possibility that it may be a matter of an "effect at a distance," of bacterial toxins which may be formed at quite different parts of the body and exhibit a pronounced affinity only to the gray substance of the spinal cord. Many problems here still await their solution.

***Flexner* and *Noguchi* have succeeded in experimentally producing the disease in monkeys by the injection of a bacterial culture.

In the fluid withdrawn by *lumbar puncture, Schultze* was the first to be able to show positive findings; he succeeded in discovering in a microscopic preparation, *Jäger-Weichselbaum's* meningo-cocci. The same discovery was made in a case of *Concetti;* in two other different cases the same author, however, found *Fränkel's* diplococci. *Looft and Dethloff* found a *Gram* stainable diplococcus, *Geirsvold* in several cases an analogous bean shaped diplo- or tetracoccus, which however could not be detected in later epidemics (*Wickmann, Harbitz and Scheel*). *Forssner and Sjövall* report finding the streptococcus citreus, or staphylococcus albus, in two cases which ended fatally. *Ellermann* found twice in the same patient, in the fluid withdrawn by lumbar puncture, amœboid homogeneous cells, which he regarded as protozoa.

Definite conclusions relative to the ætiological importance of the findings discovered so far can hardly be drawn.

Bülow-Hansen regards the intestinal canal as the *place of entry* of the infectant, while *Geirsvold* designates the pharyngeal ring (Rachenring) as such; but no sure criterion can be made about this any more, than about the manner of the spreading of the epidemic. Some authors think, that they have seen a direct transmission from one child to another (*Lundgren, Platon*), while others (*Stören*) deny this possibility. It may be regarded as proved by *Wickmann*'s painstaking epidemiological studies, that a transmission through infection by contact occurs, in which healthy persons may play an intermediary rôle. Frequently children of the same parents have been stricken simultaneously, or one shortly after the other, even in localities where, and at a time when, no epidemic existed.

Several factors of ætiological importance, especially in the sporadic cases, are surely known, to which, however, we can ascribe only the character of a predisposing or an assisting cause of the disease. We mention here *catching cold and traumatism.* Though *Oppenheim's* statement, that the latter may be regarded rather frequently as consequences of the disease, that for instance, a child falls from the chair, because the paralysis has started, may be true in many cases, but we know on the other hand, that traumatism also in other ways may act as an assisting cause in the localization of microorganisms.

On the ground of actual experience it can be stated, furthermore, that poliomyelitic paralysis sometimes follows *in the wake of other infectious diseases;* especially measles, whooping cough, scarlatina, influenza, varioloid, etc., are mentioned in this respect. I myself have seen a typical case develop after lobar pneumonia. Naturally, it can not be decided at once whether in such cases it is a matter of a secondary localization of the microorganism in the spinal cord, or whether the initial disease only prepares the ground for the specific excitant of poliomyelitis. At any rate, we wish to emphasize, that such cases are by no means frequent, and that in the great majority of cases a special ætiological factor can not be detected.

That *hereditary influences* may create a predisposition to the disease, is assumed by some from the fact, that the parents of the afflicted children were suffering from a nervous disease. Cases in which the mother during her childhood was afflicted with the same disease (*Oppenheim, Heubner*) occur, but are very rare.

Age has a great influence on the origin of the disease. By far the greatest number of cases are to be found in children during the first three years of life, while an onset after the fifth year and sporadic cases in older children and adults are observed only exceptionally; in the course of larger epidemics, however, these too are considerably endangered. An onset of the disease in the first weeks of life has been observed several times (*Duchenne* 12 days, *Baumann* 6 weeks); the intrauterine cases, which were also described, are rather questionable.

Hoche has tried to explain the frequency of the appearance of the disease in early infancy; he points to the gaping of the central canal at this age, which in later years, as a rule, exhibits extensive obliterations, and therefore is of the opinion, that the microorganism finds its way into the body by means of the central canal. "Were it merely a matter of the anatomical relations of the arterial vessels subserving the gray substance, upon which the localization of the poliomyelitis is dependent, one could hardly understand why this should not happen in the same way in adults as in children." *Lange* thought of over-exertion of the spinal cord in learning the walking and grasping motions in children, as the assistant cause.

Symptomatology and course.—The *incubation period* in epidemics does not seem to be strictly demarcated. *Cordier* found it to be in one positive case 36 hours; according to *Wickmann's* accurate statements, one may fix it at one to four days; but even longer periods seem to have occurred.

The initial stage of the disease shows mostly a febrile character, and this passes over into the stage of initial paralysis, from which develops the stage of the permanent paralysis with its secondary resultant phenomena.

The *initial stage* is rarely preceded for several days by *prodromal signs:* restlessness, weariness, loss of appetite, gritting of the teeth, premonitory tremor in the limbs, which become affected afterwards (*Neurath*).

In the majority of cases, however, the disease starts suddenly and with the greatest vehemence, occasionally over night, so that the parents find the child, which the evening before was put to bed in good spirits and healthy, paralyzed in the morning. Fever, though present in most cases, is often of short duration, lasting sometimes only a few hours, and therefore it may easily be overlooked; it may reach 101–103 degrees and in the more severe cases may last several days. In such cases other serious disturbances of the general health appear, as headache, prostration, somnolence, vomiting, more rarely diarrhœa, which, however, may dominate the whole initial stage; complete coma may also appear, and quite frequently general convulsions are observed. The later occurring paralyses are usually not to be recognized at this stage of the disease even by the most careful examination, and the presumption is certainly justified, that some cases may terminate in death even in this initial stage, and without the physician's having been able to make a correct diagnosis.

After disappearance of these stormy symptoms which, as has been said, may run their course in a few hours, and may even be entirely lacking or overlooked, the *paralyses* appear, which, *as a rule, have their greatest extension at the very beginning;* but it happens sometimes, that the paralysis in the course of the first two or three days spreads to further regions.

It is characteristic, however, that from this time on, no further progress of the paralyses can be recognized, but that on the contrary, a retrogression takes place.

In this first stage of the paralysis, several symptoms are to be found, which as a rule, disappear afterwards—namely, *pains* in the paralyzed regions, if these be handled, and spontaneous pains in the nape of the neck and the back. These phenomena probably are to be traced to a concomitant meningitis, which, according to the painstaking anatomical investigations of *Wickmann, Harbitz and Scheel*, is very frequently present; they, as well as the cutaneous hyperæsthesia, which is occasionally found, may be interpreted also as neuritic symptoms.

The same transitory character is exhibited by the disturbances in the evacuation of urine and feces, which may occasionally be present in the first week (*painful urination, retentio urinæ, incontinence*); they are to be found only when the lumbar region is affected, and therefore, are usually accompanied by paraplegia.

One of the most essential characteristics is, that disturbances in sensibility and of the function of the bladder and the rectum, are entirely lacking in the fully developed disease picture.

After the initial stage has run its course, *muscular paralysis* comes to the foreground of the phenomena. In the beginning it is extensively diffused, but retrogresses in the course of the following days and weeks, sometimes to such a degree that whole limbs, affected before, become almost normal again, so that a paraplegia or hemiplegia may be transformed into a monoplegia; in the same way, it appears that a number of muscles in the paralyzed extremities, which had lost their functions, have regained them. A complete restitutio ad integrum, it is true, occurs only in rare cases, in the abortive forms, such as are always observed in the course of epidemics. In all the other cases a part of the originally paralyzed muscle regions remains permanently impaired, and one may therefore speak with *Oppenheim* of a concentration of the paralysis to definite groups of muscles.

The great majority of cases of acute poliomyelitis lead to monoplegias (about 70%), principally to the paralysis of one leg; more rarely, only one arm is paralyzed. Paraplegia of both legs is also not uncommon, while paraplegia of both arms, crossed paralyses and hemiplegic forms belong to the rare disease pictures. Serious paralyses of all four extremities, so-called "panplegias" also occur.

The attacked muscles exhibit from the onset all the signs of *peripheral, flaccid, atrophic paralysis.* When the affected limbs are raised, they drop as if lifeless; the deep reflexes are abolished; electrical examination shows the reaction of degeneration; in a very short time, atrophy appears in the muscles.

In detail these changes are as follows: In the first days, the muscles show an increased mechanical irritability; but they are also hypersensitive to the faradic and galvanic currents, as *Sachs* was able to prove. After the first two to three days, the nerves and the muscles lose their excitability to the faradic current very rapidly, while the galvanic reaction may still remain increased for a long time. But now the well known signs of the reaction of degeneration appear, the overbalancing of the anodic excitability and the sluggish vermicular twitching of the muscle. This stage is reached even at the end of the first week. Muscles, which have once exhibited the complete reaction of degeneration, usually recover from the disease process only incompletely or not at all; if the faradic excitability is not entirely abolished, the function may be restored after the course of some weeks.

The complete loss of the deep *reflexes* is to be connected directly with the injury to the cells of the anterior horns in the spinal cord. As one leg is usually affected, the loss of the patellar reflex or a considerable decrease of it, is one of the most usual symptoms of our disease.

But it is clear without further elaboration, that the patellar reflex must be present, when the quadriceps and its anterior horn cells were entirely spared by the disease process. As a matter of fact this state of affairs exists quite frequently, and even observations of an increase in the knee reflex, have been reported (*Lövegren*, *Schüller*, *Wickmann*). They were mostly cases in which paralyses of arms existed; it is to be supposed, that a spread of the disease process to the parts of the antero-lateral column, that surround the anterior horn cause this phenomenon. Similar considerations prevail also in the Achilles tendon reflex. The cutaneous reflexes are intact or changed only in so far as they become modified by a paralysis of individual groups of muscles.

The *atrophy of the musculature* progresses very quickly. *Sachs* could recognize as early as the end of three days considerable differences in volume as compared with the sound side, and at the end of the second week, differences of 1 cm. (*Neurath*) can be observed. When the atrophy affects larger groups of muscles, naturally the entire outline of the part of the body concerned changes; the extremity may consist, as it were, merely of skin and bones. Sometimes the muscular wasting is masked by a proliferation of fatty tissue, so that one would hardly suspect, especially in very young children, the seriousness of the muscular changes.

We now take up the discussion of the paralyses in detail. Though probably all possible combinations might be occasionally observed, there are still individual types, that occur more frequently than others. It is characteristic of poliomyelitis acuta, that a whole extremity is almost never completely paralyzed; some individual muscles or groups of muscles are, as a rule, spared; besides this the very remarkable fact has been shown that frequently muscles are affected, that belong together functionally, even when they are supplied by quite different nerves. This phenomenon is probably most conspicuous in the deltoid; the clavicular portion of this muscle may be found paralyzed together with the serratus anticus major, while the middle and posterior parts are not disturbed in their function, or the latter parts are paralyzed and besides, the subscapularis (*Marie*). In the pectoral and abdominal muscles, too, similar partial paralyses of functionally related parts may be shown.

Concerning the single groups of muscles, it should be said, that in *the leg* the musculi peronei are very frequently attacked; in these cases, the tibialis anticus, as a rule, is spared. Frequently the combination of a paralysis of the quadriceps with that of the tibialis anticus occurs. It is further known, that in a paralysis of the musculature of the thigh the sartorius is almost always spared.

In the *arm*, the deltoid is usually attacked by the paralysis, frequently in combination with the biceps, brachialis internus and the supinators (arm type according to *Remak*); in contrast to this is the forearm type, in which, as a rule, the extensors are paralyzed, while the supinators are spared.

To the rarest localizations, belong the little muscles of the hand; but even such cases have been described (*Zappert and others*).

The musculature of the trunk and the back of the neck is frequently attacked. The clavicular portion of the cucullaris, on the contrary, is usually spared. The muscles of the back not rarely participate in the disease process, bringing with it especially severe disturbances of the whole equiibration of the body.

In recent years, attention has frequently been called to total or partial *paralysis of the abdominal muscles.*

In total paralysis an enormous bulging out of the whole abdomen occurs and abdominal pressure appears functionally seriously impaired.

It seems, that in recent cases, the abdominal muscles are affected mostly in a diffuse way (*Medin, Wickmann*) and that only with the retrogression of the acute stage, the partial paralyses step into the foreground. Of these, two types may be distinguished; if only parts of the transverse musculature are affected, at every deeper inspiration and every exertion of abdominal pressure, hernia-like protrusions of the abdominal wall in the region of the paralyzed muscles appear (*Ibrahim, Herrmann*) (see Fig. 192); if, on the contrary, the recti abdominis are attacked, according to the observations of *Strasburger*, a sinking of the pelvis forward is the result, combined with loss of the ability to raise one's self from a supine position without assistance from the hands. The latter type seems to be extremely rare.

Among the rarest phenomena, which nevertheless have been repeatedly observed, are *paralyses in the region of the cerebral nerves.*

The facial, especially may be affected; also the abducens, the hypoglossal and oculomotor may participate; observations by *Medin and Baginski* concerning asthmatic attacks of dyspnœa with conditions of collapse which ended lethally, point to the possibility of an affection of the nucleus of the vagus. These clinical statements agree well with the anatomic findings of *Wickmann, Harbitz, and Scheel,* who always found changes in the nuclei of the cerebral nerves.

Oculopupillary phenomena (contraction of the palpebral fissure and the pupil with synchronous retracted position of the bulbus) may be caused by an affection of the centrum ciliospinale, and have been observed in some rare cases (*Clopatt, Wickmann*).

While this stage of the initial paralysis is running its course, the *general health* of the children has long been fully restored. No trace of sickness can be found in them, other than the phenomena of paralysis. They prove to be mentally and psychically entirely unimpaired.

The only thing, besides, that deserves to be mentioned, is the finding in the *cerebro-spinal fluid.* The bacteriology of the initial stage has been already discussed. The morphology too, of the cells contained in the liquid withdrawn by spinal puncture seems not to be uniform; *Raymond and Sicard* found leucocytosis; *Brissaud-Londe, Achard, Stärcke,* lymphocytosis.

The stage of permanent paralysis develops quite gradually from that of the initial paralysis; while part of the musculature regains entirely its former function, and another recovers only partially—and an improvement in this respect may occur even after nine months or a year—part is absolutely doomed. The electric excitability disappears even for anodic irritation more and more, and may finally become entirely lost. Besides the functional loss, important secondary disturbances now appear; namely, the contractures and deformities, which are characteristic of most cases at this stage and make the patients cripples for the balance of their lives.

If all or almost all muscles of an extremity are paralyzed, no contractures appear; the limb hangs from the trunk like an appendage, that does not belong to the body—easily movable to and fro like a jointed doll.

But when, as usually happens, only single groups of muscles are affected by the paralysis, the action of the antagonists produces a *contracture* (in the sense of this effect of antagonists) quite soon, sometimes after a few weeks; at first this can be easily corrected passively, but it is gradually transformed through wasting of the tendons and fibrous degeneration of the muscles into a fixed pathological position. Besides this muscular traction, mechanical and static factors cooperate in causing these paralytic contractures. The pressure of the bed clothes, the strain upon an extremity during attempts to prop one's self up or to walk, may produce serious consequences. At the same time some other unfavorable resultant phenomena of the disease step into the foreground, namely inhibitions of growth and flaccidity of the articular ligaments. The capsular ligaments, which are frequently strengthened by tendons and are kept tense by the muscular pull may get extraordinarily loose because of the atrophy of these muscles so that the *flail-like joints* develop. Especially may such conditions occur in the shoulder and hip-joint and give rise to severe functional derangements, subluxations, etc.

Disturbances in the growth of the affected limbs almost invariably consist of an inhibition of growth, and this may be quite considerable. The paralyzed extremity is shorter (up to 20 cm.), the bones as shown by the X-ray diminished in thickness and osteoporotic; in addition there is a marked disposition to fractures. The skin is pale or cyanotic, and feels much cooler to the touch, than that of the sound side; the difference in temperature may amount to several degrees. In rare cases the skin shows trophic changes, hard edema (*Oppenheim*) or disturbances in hairgrowth and secretion of sweat, anhidrosis (*Higier*). Diminution of the pulse, too, may appear in the side with the arrested growth. In some rare cases a lengthening of the diseased limbs was noted (*Seeligmüller*, *Neurath*, *Oppenheim*).

The atrophic paralyses of the muscles of the back may give rise to the most severe scolioses and lordoses.

Deformities produced by the paralytic contractures are especially frequent, and especially annoying in the foot and leg. According to the type of paralysis there develops a pes planus, pes varus, or equinovarus and pes calcaneus. The deformity may be so extensive that the patients are forced to walk on the dorsum of the foot. Genu recurvatum and incurvatum, too, may appear as sequelæ of poliomyelitic paralyses.

If the quadriceps be atrophied or should this condition be combined with serious paralyses of the muscles of the back, the children are often able only to walk on all fours with the help of their hands (Fig. 200). In such unfortunate creatures an occupational hypertrophy of the arms can occasionally be observed. It is one of the greatest triumphs of orthopædic surgery to have enabled a number of such handwalkers to again walk upon their legs (*Vulpius and others*).

However badly the physical and not seldom indirectly also the mental conditions of the patient are impaired through the deformities described above, life itself is not endangered, even in the most serious cases, since the paralysis is never progressive. Still it should be mentioned that a few cases of *recurrence* of the disease have been reported (*Marie*, *Wendenburg*) and that cases have been observed also, in which in later years a progressive muscular atrophy appeared. "It seems, as if the cells of the anterior horns, having been damaged once by the acute disease, even though it has been recovered from, were later on not sufficiently well developed to meet the expected functional requirements and therefore become atrophied prematurely" (*Strümpell*).

Pathological Anatomy.—While the first investigators of the diseased spinal cord observed only the processes, that had long before run their course, in the last two decades a large number of recent cases have been studied, which enables us now to form a rather exact picture of the pathological histology of the initial stage. Besides the celebrated first cases of this kind of *Rissler*, *Dauber*, *Goldscheider*, *Mathes*, it has been especially the most recent investigations of observers (*Wickmann*, *Lövegren*, *Harbitz and Scheel*), that have given us detailed information in this respect. We know, that in the macroscopic study only a hemorrhagic focal change of the anterior horns is seen and this may even reach softening, but the meninges show neither opaqueness nor exudation.

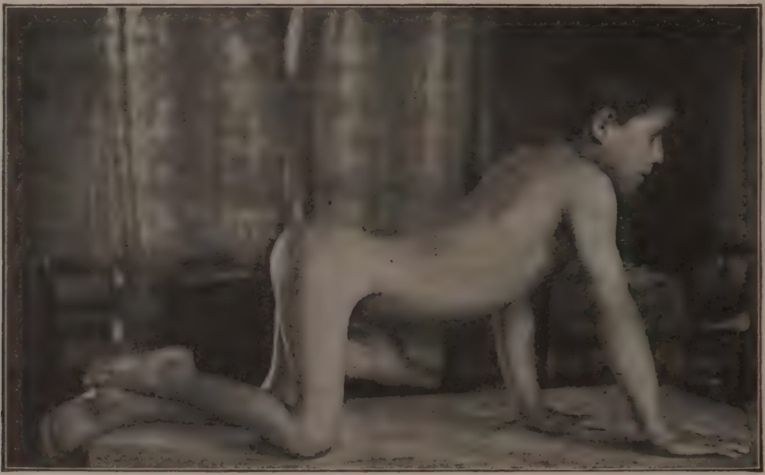

FIG. 200.—Infantile spinal paralysis (hand walker). (Observation of Prof. O. Vulpius, Heidelberg.)

In the microscopic investigation severe inflammatory changes are found in the diseased regions. The vessels are dilated and proliferated, in part thrombotic. Extravasation of blood into the tissue can be seen. In the vicinity of the vessels accumulations of round cells can be found. The ganglion cells are seen to be badly damaged, cloudy and swollen, the processes shrunken. The phenomenon of neuronophagia, too, has been seen frequently in recent times. The inflammatory process is to its greatest extent localized in the gray substance of the anterior horns, without, however, being absolutely limited to the same, as it encroaches not only on other parts of the gray substance (in the upper lumbar and the lower dorsal portion of the spinal cord especially in *Clarke's* columns), but as a rule also on parts of the white substance. The process is invariably far more extensive, than one would conjecture from the clinical symptoms. Though the cervical and lumbar enlargements are attacked preferably and with greater intensity, still very large regions of the entire spinal cord are always affected, and in all cases, in which the investigation was extended further, observers found analogous changes in the medulla oblongata and the cerebral cortex. Microscopically frequently an inflammation of the meninges was recognizable, a finding which was emphasized already by *Schultze*, and to which *Harbitz and Scheel* ascribe a special importance. A paramount dependence of these processes upon the arteria centralis, which was accepted by many authors (*Goldscheider*), does not exist according to more recent investigations (*Wickmann*). The theory of *Charcot*, which was later defended by *v. Kahlden* and more recently again taken up by *Lövegren*, that the process is initiated by a focal degeneration of ganglion cells is hardly probable, as the interstitial inflammation not only controls the picture, but exists with normal ganglion cells (*Goldscheider*, *Wickmann*). The degeneration of the nerve cells is most likely a consequence of the inflammation and may be even a coordinate phenomenon of it (*Medin*, *Schwalbe*, *Neurath*). Recently *Heubner* reported a case, in which degenerative changes of the ganglion cells were found exclusively without any inflammatory phenomena.

In the later stages of the disease the foci are found to be sclerotic and atrophic. The ganglion cells have disappeared or show a grave degeneration; cystic spaces may indicate the former localization of the disease. The walls of the blood-vessels show thickening, the glia shows proliferation; the entire half of the spinal cord may exhibit, even macroscopically, a visible diminution, the anterior horn appears greatly narrowed, the demarcation between anterior horn and white substance blurred (Fig. 201).

The anterior roots and nerve trunks corresponding to the disease foci are atrophic. The muscles show degenerative changes. During operations the opportunity is frequently given to observe with the naked eye the most diversified stages adjacent to each other. Instead of the normal, deep red color of the muscles there appears a salmon-red, light pink, grayish-red or even yellowish or whitish-yellow tint; the muscles may sometimes even have almost entirely disappeared. It happens frequently that in a muscle degenerated parts can be found adjacent to sound parts, in which case the muscle shows a tiger-striped appearance (*Vulpius*). Upon microscopic examination the individual muscle fibres are seen to be narrowed, split asunder, vacuolated and infiltrated with fatty granules; the transverse stripes are lost, the nuclei of the sarcolemma are increased in number. At the same time, however, hypertrophic muscle fibres are also present quite frequently but they exhibit degenerative phenomena also.

Nosological Position of Infantile Spinal Paralysis.—While our disease has been regarded in the past as a clinically and pathologically well-defined disease entity, now, considering the more extensive pathologic-anatomical investigations and the facts obtained by the observation of epidemics, the conviction is growing more and more, that acute poliomyelitis is really only a clinical conception, and with regard to its ætiology and its patho-histology ought to be regarded as a member of a larger group of diseases, for which *Wickmann* has suggested the name *Heine-Medin's disease.* All the nervous diseases, that are observed in common with poliomyelitis in epidemics and probably owe their different clinical picture to a different localization of one and the same disease process (and micro-organism?) would have to be classified in this group. *Wickmann* from this point of view describes eight different forms of the *Heine-Medin* disease: 1, the spinal poliomyelitic; 2, that which exhibits the picture of an ascending or descending paralysis (*Landry's* paralysis); 3, the bulbar (*Medin*) or pontine (*Oppenheim*) form; 4, the cerebral encephalitic; 5, the ataxic; 6, the polyneuritic; 7, the meningitic, and 8, the abortive forms. There is no denying the justification of this far-sighted point of view, but practical reasons make it advisable in our opinion to retain the clinical conception of infantile spinal paralysis.

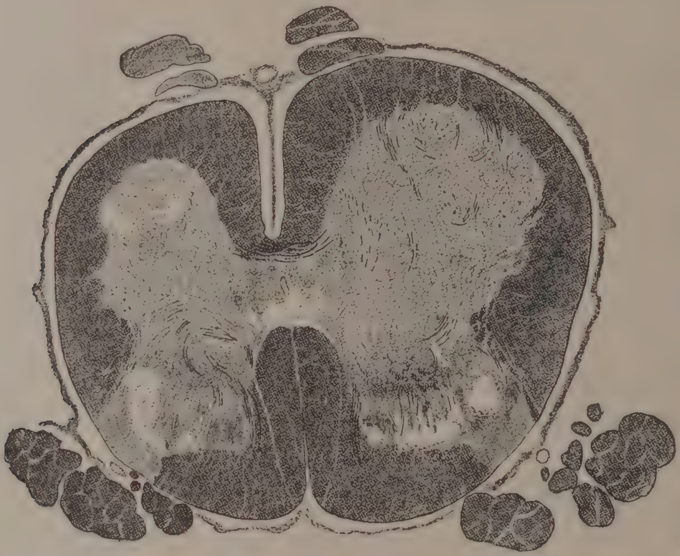

FIG. 201.—Poliomyelitis acuta having run its course. Section from the lumbar cord; staining according to Weigert. Narrowing of the left half of the spinal cord section; atrophy and sclerosis of the anterior horn; degeneration of the anterior roots at the left side. (*From Schmaus.*)

Diagnosis.—In the initial stage a diagnosis of acute poliomyelitis will be possible, as a rule, only in very rare cases, and then only if the special suspicion of the physician is directed to this disease through the epidemic accumulation of the cases. Not until the appearance of the paralysis is the recognition of the disease ordinarily possible. The fact that the acutely beginning paralysis does not exhibit a progressive tendency, that on the contrary the maximum extent of the paralyses exist in the beginning of the disease, further the flaccid atrophic character of the paralysis, the absence of disturbances in sensibility and in the sphincters of the bladder and rectum are characteristic and of diagnostic importance for spinal infantile paralysis. Because of these symptoms, diagnosis is usually not difficult, but differential diagnosis may meet with great difficulties in specially conditioned cases.

Differential Diagnosis.—*Multiple neuritis* may induce quite similar clinical pictures, but it is quite rare in early childhood. The following considerations make a distinction possible: Multiple neuritis in contradistinction to poliomyelitis acuta develops its complete clinical picture but slowly, in the course of days and even weeks; it exhibits therefore, from the first, a progressive character; the febrile stage is longer in neuritis and fever may return from time to time. Disturbances in sensibility in neuritis are present, but in small children not always readily demonstrable. Painfulness in the muscles and nerve trunks points to neuritis only in the later stages; in the initial stage it is also quite frequent in poliomyelitis. Edema and the participation of cerebral nerves as well as ataxia speak rather for neuritis; the same is true of paralysis, in which the muscles attacked correspond strictly to the peripheral innervation, while poliomyelitis shows a predilection for muscles which are functionally related. An onset with convulsions is said never to occur in polyneuritis.

Hæmatomyelia after traumatism is distinguished from poliomyelitis with difficulty, in cases in which disturbances in sensibility are lacking.

Acute myelitis, which is rare in childhood, is to be distinguished from pure poliomyelitis through phenomena, which point to an affection of the white substance (disturbances in sensibility, spasticity, ataxia, participation of the sphincters).

Infantile cerebral paralysis may produce difficulties in some cases; for instance, if the poliomyelitis be associated with paralysis of cerebral nerves. But the detection of a flaccid degenerative character of the paralysis, of the decrease in the tendon reflexes, the reaction of degeneration in the muscles, eventually the monoplegic type of paralysis, prevent confusion; the acute onset of both diseases may show a quite similar course, and combinations of both diseases may also occur. It should be mentioned again, that the absence of the patellar reflex, though characteristic for poliomyelitis, is by no means an obligatory symptom, and that even exaggeration of the knee reflexes may occur in infantile spinal paralysis. If in the further course of the disease, convulsive seizures, epilepsy, mental and psychical impairment appear, these point to an infantile cerebral paralysis, as do also athetosis and the presence of a unilateral *Babinski* toe phenomenon in the paretic half of the body in older children. Examination with the faradic current enables one to make a definite decision in doubtful cases.

Obstetric paralysis may resemble acute poliomyelitis in every respect. Inward rotation of the arm, because of the paralysis of the infra-spinatus, speaks for obstetric paralysis; the anamnesis is decisive.

Congenital muscular defects may in some cases be distinguished through synchronous malformations of a different kind, for instance, webbed fingers or toes (*Oppenheim*).

Myatonia congenita (*Oppenheim*) may produce difficulties in its demarcation from infantile spinal paralysis, but only to those who have not previously seen a case. A careful study and an electrical examination, which only rarely shows the presence of reaction of degeneration, will prevent a mistake in this respect.

Inhibition paralyses (*Vierordt*) in rachitics and children affected by hereditary syphilis, may also resemble poliomyelitis; but close observation will always prove, that it is a matter only of pseudoparalyses; the electrical findings besides, are not abnormal.

Also the *paralysie douloureuse*, due to traction upon the arm, is easily recognizable as a pseudo-paralysis, and can also be distinguished by its rapid recovery.

Hysteric monoplegias, which occasionally may be accompanied by pronounced atrophy, can be recognized upon anamnesic grounds, and by the normal electrical findings.

Progressive muscular dystrophy may be concerned in differential-diagnostic respect, and even regarded as poliomyelitis acuta, if the anamnesis is lacking; the distinction may then be only feasible through prolonged observation; this is the case not only with spinal muscular atrophy (*Werdning–Hoffmann*), but also with *Hoffmann's* neural muscular atrophy, which attacks older children only and sets in with an atrophy of the peroneal group of muscles. The bilateral symmetry and the slow progress militate against poliomyelitis acuta; besides, in the latter, the reactions of degeneration are usually far more pronounced.

Tumors of the spinal cord, spondylitis, are always accompanied by disturbances in sensibility and in the sphincters, and by an increase of the reflexes. The reaction of degeneration is lacking, as is also the acute onset.

Post-diphtheritic paralyses may lead to a confusion with poliomyelitis, especially in smaller children who can not give an account of their disturbances in sensibility. The slight atrophy and the rapid restitution, eventually, the gradual spread of the paralysis, prevent this confusion, even if the primary affection is not known.

Paralyses of the legs in spina bifida (*occulta*) are combined with paralyses of the sphincters and disturbances in sensibility.

Paralysis of abdominal muscles in poliomyelitis acuta has frequently been mistaken for genuine abdominal hernias. The proof of other paralytic residues, and possibly the localization of the protruding tumor, will prevent a mistake in diagnosis.

Prognosis.—Life itself, as far as our knowledge goes, is only rarely threatened by acute poliomyelitis. In epidemics, according to the reports of *Wickmann*, an exitus letalis is more frequent, especially in older children and adults, than is generally assumed to be the case. As to the younger children, the prognosis is more favorable, in so far as total recoveries are by no means infrequent.

In general, however, complete restitution is very rare. Usually, even in the lighter cases, some defect remains. Especially ominous are the grave paraplegias of the legs, which may permanently rob the patients of their ability to walk; the paralyses of the trunk also are to be regarded as belonging to the most unfavorable localizations. If only one leg is paralyzed, modern orthopædics almost invariably succeeds in restoring the ability to walk. Even the paralyses of the shoulder, which were formerly regarded as so very unfavorable, because they rendered the entire arm and hand functionally useless, offer a more favorable outlook, if arthrodesis be performed.

In the individual case, it is very difficult to state during the first few days to what an extent the paralysis, which has appeared, is capable of restitution. Generally speaking, less of the functional disturbance will remain if from the beginning only a small region was attacked by the paralysis. As to the individual muscles, the electrical examination affords somewhat reliable conclusions. Muscles which after the course of two or three weeks can still be contracted by the faradic current will regain their function. Where complete reaction of degeneration can be observed, the hope of a restitution is but very slight. But a partial recovery is even possible then.

Spontaneous improvements occur even after six to nine months. After a year has passed, the disease process may be regarded as finished.

In every case of spinal infantile paralysis, in which the diagnosis has been definitely made, the parents of the patient may be assured most definitely that the mental development of the child will suffer in no way from the nervous affection.

Treatment.—In the onset of the disease during the febrile initial period complete rest is the most important consideration. The fever can be combated with phenacetin or aspirin and diaphoretic measures, which may be effectively furthered by hot-air baths or dry pack, and by hot drinks that make the patient perspire. Baths, on the contrary, are to be avoided in the interests of rest. At the same time, one will take care that there is regular evacuation of the bowels and order a bland, non-irritating diet, strictly avoiding all beverages that contain alcohol or caffein.

To bring about a derivative effect directly on the disease focus, different means are recommended (inunction of unguentum cinereum 0.3–0.5 several times a day, an ice coil or ice bag, a blistering plaster, painting with tincture of iodine). But it would seem more advisable to omit everything that produces an unnatural position in the patient or is liable to prepare a foundation for the origin of a decubitus or inflammations of the skin. It would be most useful, probably, to bleed the patient once in the region of the back at the level of the presumed disease focus, and to perform a lumbar puncture, which according to all theoretical implications can be conducive of nothing but good. *A. Starr* recommends giving urotropin in the initial stage, which, in his opinion, passes into the cerebrospinal fluid in the form of formaldehyde. Washing and rubbing the skin of the back with spirits may also be made use of in the initial stage, especially when quite extensive paralyses prepare us for a long bedridden stage.

Even in the slightest cases, the patients must be kept in bed absolutely 2 to 3 weeks. After the first 2 weeks, one may commence with local *mechanical therapy* of the musculature, which must be continued without interruption as long as hope is still justified relative to further restitution of the functions. This treatment by massage and electricity, which must be continued for many months and even a full year, greatly taxes the patience of the physician and the patient; but it is a fact not to be denied that with great care and especially with scientifically applied massage, the recovery and strengthening of the muscles takes place much more quickly and completely, than by letting the paralyzed muscles alone.

Electricity is applied in such a way that one uses currents of a strength and quality which, according to the existing stage of the paralysis, just suffice to excite twitchings. The application of the faradic current is therefore of value only when the muscles can still be excited by it. As a rule, the galvanic current will have to be used, the cathode, in severe paralyses, being usually placed on an indifferent spot (or upon the back at the level of the principal focus) and the paralyzed muscles stroked with the anode, constantly increasing and diminishing the current, or by exciting slight twitchings through opening and closing the current. The seances, which should last about 10 minutes, should be repeated at first daily, and later every other day; if the improvement continues, the cathodic current may be tried, and later still the faradic current be resorted to. Electrization of the spinal cord itself is not advisable. In all manipulations with wet electrodes, particular care must be taken to prevent the little patients from getting too wet and catching cold. Such a danger is imminent and very disagreeable; complications may arise therefrom.

After each period of 4–8 weeks, the electrical treatment should be discontinued for 1–2 weeks to prevent a nervous irritation of the patient (*Eichhorst*).

Massage should be applied once or twice daily in short seances and in case the paralyses be more extensive and the patients bedridden, should be applied also to the sound, but inactive musculature. Massage combined with active and passive movements is by far the most important therapeutic measure to effect favorable results and prevent the formation of contractures.

After the lapse of the first weeks besides massage and electricity, *warm baths* (96 to 101° F.) have proved useful, to which may be added aromatic ingredients or brine (2 to 3 pounds of ordinary salt to each bath).

In serious cases great care should be taken to place the patient in an appropriate position to prevent static or mechanic contractures or deformities. The children ought not to lie in bed with their legs drawn up on the abdomen and pressed together. The feet, which are especially endangered, must be protected from pressure of the bed-clothes by a wire frame.

Not too much should be expected from such *internal medication* as has been recommended to incite absorption or to combat the inflammatory phenomena in the spinal cord (preparations of iodine, ergotin); possibly subcutaneous injections of strychnine might be more effective.

In later months on the contrary, a judicious strengthening of the general condition of the patient through appropriate feeding and eventually through sending the patient to the country or the mountains will be of special importance.

Also *hydrotherapeutic treatment* at Nauheim, Toelz, Kreuznach, etc., is said to be very efficacious.

The muscles, which have partly recovered from the paralysis, ought to be kept from inactivity by exercising, by motions against resistance (possibly with an appropriate apparatus), as a relapse into the paralytic state is only too likely to occur, when the extremities have not become at once perfectly usable.

Any remaining deformities may frequently still be capable of the most splendid restitutions by *orthopædic methods.* Here, too, timely treatment is indicated, but it seems to me that the important rule ought to be observed, not to undertake any operative interference before the period of spontaneous restitutions is well over, namely not before 6 to 9 months have passed.

While one was formerly restricted to *tenotomies and redressments*, modern orthopædics has quite a series of methods at its disposal of which the right selection must be made according to each individual case, but which may be combined too, and so made to produce astonishing functional results. I can not enter into these matters here more thoroughly, hence deal briefly with the principal ones only. Many patients owe to *portable orthopædic apparatus* a considerable improvement in their condition. These machines serve above all to fix loose joints, furthermore to regulate the motions of partially paralyzed joints; they may be of use also for the correction of faulty position of the joints. But they have the great disadvantage, that the paralyzed limbs have to carry the weight of the apparatus and that a functional curative effect can not be brought about by their application. One should not expect much of the much vaunted rubber pullers (artificial muscles).

A procedure, which may be very serviceable in many cases is *arthrodesis* (*Albert*), the operative stiffening of a joint; it is to be considered principally for the ankle-, knee- and shoulder-joints, if all or the greater part of the muscles that move these joints are destroyed. By this operation the ability to walk may be restored and a lame arm, whose forearm and hand musculature is preserved, but which hangs from its loose shoulder joint like a useless appendage may recover its full usefulness.

The third procedure is the *transplantation of tendons*, first introduced by *Nicoladoni*. Muscles, that have remained sound or parts of such, are used to replace lost functions, in that they either are made to unite with the peripheral tendons of the degenerated muscles or that a transplantation of the muscular pull to the most appropriate point of insertion upon the bone is brought about by artificial tendons made of silk (*Lange*). It has been proved, that not only muscles belonging to the same functional groups but also antagonists may be used to effect such a transplantation, so that for instance the paralyzed quadriceps femoris may be replaced by the flexor of the leg proper.

We can not enter here into the details of the technic, which must be thoroughly mastered to insure success. The exact plan of operation, the decision as to which muscles must be regarded as sound enough to act as sources of power and how in the individual case, by taking into consideration exactly what is lost and what is still retained, the most favorable result possible may be obtained, is an art in itself. According to the experience of orthopædists the macroscopic inspection of the muscles before the operation is sometimes more important for judging their functional ability, than the electrical examination, which, however, naturally should always be employed as an aid.

Concerning another method, that may prove successful in the future, *nerve grafting* as applied to infantile spinal paralysis, only very few reports have been made known (*Hackenbruch*), so that its mere mention here must be regarded as sufficient.

VII

HYPERKINETIC DISEASES

BY

FR. PINELES (Vienna)

1. CHOREA

(a) Infectious Chorea

Infectious chorea (or *chorea minor*,[1] *Sydenham's chorea*, *St. Vitus dance*) is a disease of the brain, brought about by an infectious agent and characterized by involuntary and incoordinated movements, as well as by changes in mental state.

Symptomatology.—Usually, the first symptoms appear in rather arbitrary fashion. The patients become apathetic, peevish, irritable and exhibit gradually increasing general *restlessness* and *awkwardness* in the performance of even simple movements which becomes more and more noticeable. Thus they seize an object with an uncertain grasp, or let it fall, and are unable to stand quietly, to sit, write, knit, etc. It is visibly an exertion for them to remain quiet for even a short time, and this very exertion leads quite as often to the opposite goal, since the effort degenerates into choreic twitchings. With these ambiguous symptoms, it is not strange, that the people around are mistaken as to the disease, and try to correct it, as misbehavior, by pedagogic means.

Soon the real muscular twitchings begin to appear, first in the *hands*, *arms and face: purposeless*, *involuntary movements*, which either appear spontaneously in a state of rest or interrupt an intended movement. Other muscles also, like *those of the eyes and tongue and those employed in deglutition and respiration* succumb to these twitchings; the legs are affected usually only at a later stage and to a slighter degree. All these twitchings are of short duration, like quick, brusque, voluntary movements, from which, however, they are distinguished by their *lack of system*, *purpose and skill;* also by their irregular appearance which causes them to be seen now here, and now there; the patients display a muscular restlessness, which recalls the gesticulations of marionettes, that are moved at will upon the stage.

Upon observing such a patient, a number of striking phenomena attract our attention. The head is thrown, now to one side, now to the other, now backwards. *In the face*, the twitchings produce lively grimaces; the forehead is wrinkled, the eyebrows contracted, the mouth drawn to the right or to the left, then closed, then opened again, as if the patient were snapping at something. At the same time clucking, smacking, or exulting cries are often heard. If the *tongue* is protruded, it is rapidly thrust out, only to be just as rapidly pulled back again. The *hands* are alternately flexed and extended, the fingers spread apart, the arms rotated inwards, the shoulders drawn up. The *legs* are now extended, now pressed against each other, then again rotated outwards. *Speech* is impaired through the choreic restlessness of the tongue, glottis and abdominal muscles (*v. Ziemssen*), and since the movements of respiration occur by fits and starts, the patients utter only a few syllables, while by involuntary quick and deep inspirations they interrupt themselves in talking. If the *respiratory muscles* are involved, the intervals between the respirations are irregular. The movements of the *muscles of deglutition* may be disturbed to such an extent, that taking nourishment is interfered with. *Gait and writing* become erratic and irregular. In sleep the choreic twitchings cease. With the intensity and spread of the disease the entire body finally succumbs to muscular twitchings, so that the patients can neither stand nor walk. When in bed, they are often bathed in perspiration, tossed here and there, or hurled upward; indeed, in the most severe forms of the disease, they even throw themselves out of bed. With laboring, panting respiration, and gradual loss of consciousness, general loss of strength and cardiac weakness may *lead to death.*

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[1] Chorea major belongs to the disease picture of hysteria.

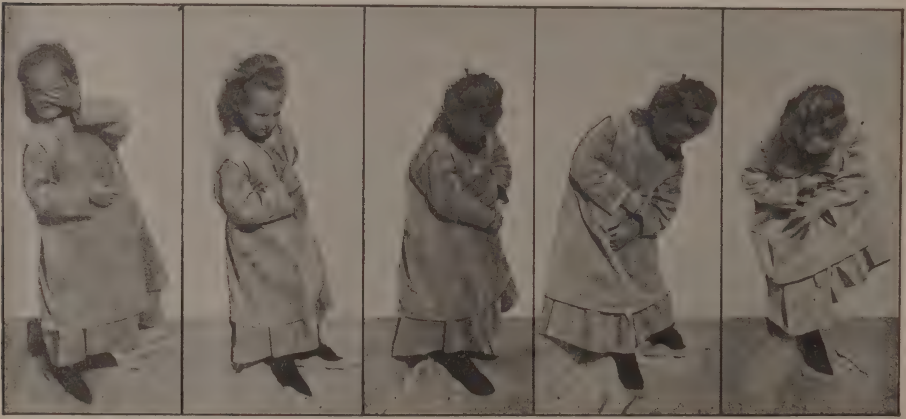

FIG. 202.—Chorea minor. (Command to stand still.) (Medical clinic, Heidelberg.)

In many cases, especially at the beginning of the disease, only one-half of the body is attacked (*hemichorea*) or more severely involved. Rarely *motor weakness* predominates from the beginning of the disease, the choreic twitchings not being severe. But even then we can convince ourselves by close examination, that only a *pseudoparesis* (*Oppenheim*) is present, since the limbs, which hang down limply, can nevertheless still perform various movements (so-called chorea paralytica). The *electric excitability* of the nerves and muscles is normal, the *reflexes* mostly unchanged, the *function of the bladder and bowels*, intact. In some cases there is increase in temperature, in exacerbation of the disease process, temperatures up to and even above 104 may occur.

Besides these physical phenomena, the *psychical* changes play the most important rôle. All sorts of transitions from slight to the more pronounced depressions are found. The patients are extremely irritable, easily frightened, whimsical, moody, evince on the whole an unstable disposition, sudden change of mood, inability to collect themselves, but without any disturbance in intellect, which, on the contrary, frequently shows great mental keenness. Only in a few cases—and those the most severe symptomatically—is there any *mental disturbance*, especially in older persons, or in those at least more than 15 years old, which presents the picture of intoxication deliria, with high degree of confusion, hallucinations, stuporous conditions and fits of rage (*Möbius*).

In *post-hemiplegic* chorea, a comparatively rare disease picture, one observes swinging, rotating and shaking movements, which in connection with incomplete hemiplegias appear in the paretic limbs, more frequently in the arm than in the leg; only exceptionally are the muscles of the face, palate, and tongue affected. Often violent pains are felt in the affected limbs; furthermore, the choreic twitchings are often combined with tremor and athetotic movements. During voluntary movement, there is an increase in the twitchings. The affected muscular groups are usually relaxed, hypotonic. Post-hemiplegic chorea is found in disease processes, which are located in the posterior part of the thalamus, the red nucleus and the region of the superior cerebellar peduncles.

Etiology and Pathogenesis.—Chorea infectiosa appears usually in later childhood, most frequently between the 7th and 13th years and prefers with respect to frequency and intensity the female sex, in whom, when it appears after the 15th year, it occurs almost exclusively. The proportion between the two sexes is about 3 to 1. But the disease, though more rarely, occurs in every period of life, even in extreme old age (*chorea senilis*). It is met with more frequently in the cool and moist, than in the warm and dry seasons of the year. *Heredity* is very rarely demonstrable in acute chorea; on the other hand, it is striking, that sufferers from this disease often have a *neuropathic predisposition*. It is mostly sensitive, anæmic, excitable individuals, who have a tendency towards the disease. The relations of chorea to *pregnancy* are of importance, wherein it must be emphasized that young women, women bearing children for the first time, and women during the first half of pregnancy are especially predisposed; chorea but rarely appears during the puerperium.

Among the statements made by patients, it is found rather often, that *psychic excitement and fright* are assigned as the cause of the disease. But since such occurrences are far more frequent, than the phenomena supposedly resulting from them, one is apt to think sometimes, that too much attention should not be paid to them; nevertheless, one must not exclude the possibility, that fright and excitement may play their part among the purely psychic causes of the disease, as an excitant factor.

Among the causes of chorea, *acute articular rheumatism and endocarditis* are undoubtedly of the greatest importance. All three diseases occur in all possible interchanging relations. Thus, extensive statistical investigations (by *Ziemssen*, *Sée*, *Mackenzie*, *Gowers*, *Starr*, etc.) have shown, that in at least one-third of the cases of infectious chorea the patients formerly suffered from polyarthritis acuta, which in turn was frequently succeeded by endocarditis. Furthermore, one sees acute rheumatism of the joints and endocarditis develop during the course of chorea, or finds in choreic patients the symptoms of a cardiac defect. The chorea of persons, who have advanced beyond the age of childhood and the chorea of pregnancy are most frequently accompanied by a cardiac defect. In some few cases, chorea appears in connection with other infectious diseases, like scarlatina, measles, and then its course is less favorable. All these facts, taken in connection with other considerations, indicate that in dealing with chorea minor, we are dealing with an *infectious disease*. Such considerations are: the usually sudden appearance of the phenomena of the disease, the rather typical course, the preference for a certain period of life, the usually typical disappearance of the picture, the strong tendency to relapses, the acute mental disturbances, that bear the stamp of the toxic deliria, which appear in the train of acute infectious diseases. From these preliminary statements, it seems probable that the "chorea virus" stands in close connection with the virus of acute articular rheumatism. As to the pathogenetic details of chorea, we have, as yet, no clear conception. Against the hypothesis that coagula, loosened from the cardiac valve affected, lead to chorea through occlusion of the most delicate vessels of the brain (*Kirkes*, *H. Jackson*, *Frerichs*, *and others*), the findings reported present in many cases—the frequent absence of anatomic changes in the brain—must be taken into consideration. Just how far the view of *Bonhöffer*, that at the basis of the choreic disturbance in motility lies a centripetal functional disturbance, due to a lesion of the cerebellar-peduncular tract (or its continuation in the subcortical ganglia), can be applied to infectious chorea, can not be decided at present.

Course and Prognosis.—Infectious chorea is, on the whole, a *benign* disease, only 2–4% of the cases ending fatally. The usual course is completed in 2–3 months, the average being 10 weeks (*Gowers*, *Oppenheim*); occasionally the disease process is protracted, especially in persons no longer young, where after improvement of the phenomena, a recurrence of the process follows (chorea adultorum permanens). In some cases, very much reduced, physically or mentally weak children display from the beginning a very confused disease picture, which only disappears after a year or more. The tendency to *relapses* in infectious chorea is very strong; at least one-fourth of all cases are attacked several times. In most instances there are two or three attacks, but their number may be increased even up to ten (*Sachs*, *Gowers*). With the increase in relapses the cardiac affections also become more frequent.

The **prognosis** in children and the young is usually favorable. It is most unfavorable in the wake of scarlet fever, measles, and typhoid, as well as in those cases in which a severe disease picture develops (chorea acutissima). In grown persons it is probably less favorable, because of the endocarditis, which appears more frequently and with greater severity at this age. The prognostically most dangerous form of acute chorea is chorea gravidarum, in which the mortality fluctuates between 20 and 30% (*Fehling*, *Gowers*, *Oppenheim*). Death occurs as a result of general exhaustion, insufficient nutrition and lack of sleep.

Pathological Anatomy.—The anatomical substratum of infectious chorea is *not known*. Autopsy of the cases, that resulted in death at an acute stage of the disease, frequently revealed delicate, small, *endocarditic vegetations* on the mitral valve (*Kirkes*, *Ogle*, *Tuckwell*, *Pye-Smith and others*), sometimes also a vitium cordis or a fatty degeneration of the myocardium. The findings in the brain present, as a rule, nothing striking. Occasionally *embolic obliterations* of larger and smaller vessels of the brain and inflammatory processes about them are met with. The small light refracting granules, described as "*chorea particles*" *by Elischer and Jakowenko*, and which are found in the lenticular nucleus, have been connected with this disease, but as they are found in other diseases also, they can not be considered at all characteristic of chorea (*Wollenberg*).

Differential Diagnosis.—In by far the majority of cases, the diagnosis of chorea may be made with certainty, but occasionally great difficulties are met with. Thus *hysteria* may present a disease picture very much like that of chorea. It is then more frequently a question of epidemics in schools, where through imitation the children are attacked by choreic twitchings. In such cases possible sources of imitation should be looked for, also hysterical stigmata and under certain conditions the therapeutic effect of hypnosis must be tested. It must also be remembered that hysteria with choreiform twitchings may attach itself to an infectious chorea. Occasionally it is difficult to distinguish tic from chorea. In the former disease the twitchings are of the nature of purposive coordinated movements, affect preferably the large joints, appear intermittently and are very often combined with obsessions and coprolalia. It is also possible that *infantile cerebral paralysis*, should it be accompanied by choreic twitchings, may lead to confusion with chorea.

For infantile paralysis are characteristic: rigor in the muscles attacked by choreic restlessness, increase in the tendon reflexes, and the combination with athetoid movements. If hereditary *degenerative chorea* appears at a relatively early age, and direct heredity is not known, only long-continued observation can prove whether an infectious or a degenerative chorea is present.

Treatment.—The greatest stress must be laid upon absolute *physical and mental rest.* In every case the child must be forbidden to attend school for a twofold reason, since there is always danger that the other children may develop an hysteric chorea from imitation. In very slight degrees of the disease, the patient should stay in airy rooms, may even move about a little in the open; in forms of medium severity, *rest in bed* is absolutely necessary. If there be danger of injury from the violent muscular restlessness, a *bed* should be prepared with a soft *mattress* and the walls lined with cushions or mattresses. The padded so-called "*spasm-beds*" are also very well adapted to chorea patients. Should a *psychosis* supervene, the patient must be transferred to a proper institution. The diet should be lacto-vegetable, with abstinence from coffee, tea and alcoholic liquors.

Arsenic is regarded as the most reliable medicinal agent. Daily after dinner and supper the patient should be given three drops of liquor arsenicalis Fowleri diluted with distilled water; the dose for small children should be gradually increased until it reaches twelve drops twice a day, for young persons until it reaches twenty drops at each dose, then it should be slowly returned to the original amount. Acidum arsenicosum (1/2 to 3 mg. daily) also has a very good effect. Arsenic is nearly always well borne by choreic patients and may be given for 2–3 months. Naturally, it must be discontinued upon the appearance of symptoms of poisoning, (diarrhœa, conjunctivitis, herpes). As antirheumatic medicines, we would recommend natrium salicylicum (three times daily, 0.5 to 1.0), antipyrin (twice daily 0.3) or salipyrin (twice daily 0.5). In conditions of excitement and the severe forms, bromides (natr. bromat. 2.0 to 5.0 daily) are useful. Treatment of the very severe cases of chorea, in which sleep and nourishment are very insufficient, is very difficult. Of soporifics the best are: subcutaneous *injections of morphine, amyl hydrate* (3.0 to 4.0 per rectum), *veronal* (0.2 to 0.4) and very light *inhalations of chloroform.* The latter is to be considered especially, should it become necessary to feed the restless patients by means of a tube. But in the repeated use of chloroform, one must not forget its evil effect upon the heart. For the same reason, chloral hydrate is absolutely contra-indicated. Prolonged lukewarm as well as *hot baths* often have a very quieting effect, while the effect of the galvanic current is very doubtful.

If in *chorea gravidarum*, the choreic twitchings appear in considerable intensity, if the patients are very much reduced, and any severe complications (like vitium, nephritis) are present, the advisability of *artificial premature delivery* should be considered.

(b) Degenerative Chorea

Degenerative chorea (also known as hereditary, chronic progressive or *Huntington's* chorea) is a rare chronic hereditary disease, which attacks chiefly *adults* and is characterized by *disturbances of the intellect* in addition to the choreic movements.

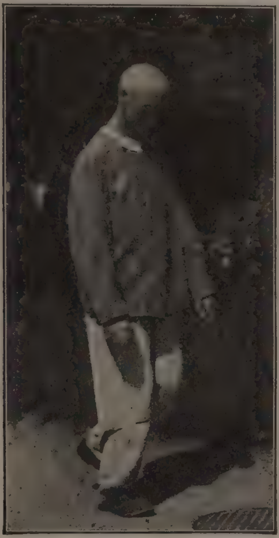
FIG. 203.

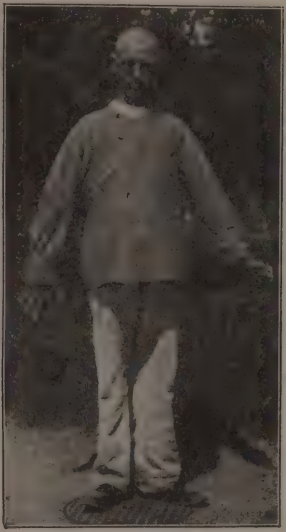
FIG. 204.

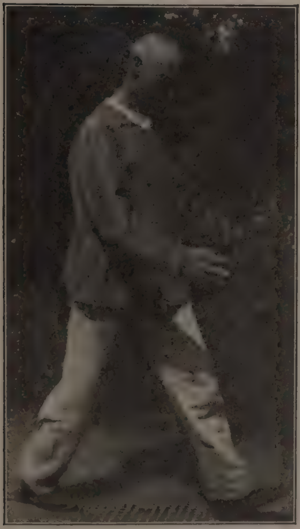
FIG. 205.

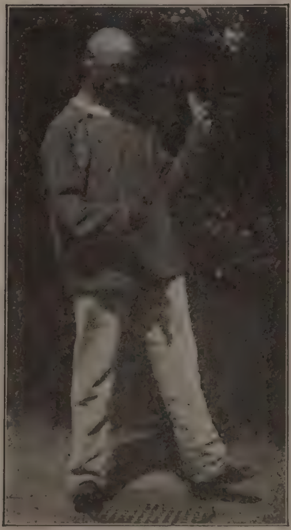
FIG. 206.

FIGS. 203 to 206.—Grave case of chorea chron. hered. (Huntington) with pathetic gesticulations, disturbance in gait, etc. (Medical clinic, Heidelberg.)

Etiology.—The disease is transmitted by *direct heredity* from one generation to another; but there are in these "choreic families" also individuals, who remain quite healthy, and in such cases, their posterity is nearly always free from chorea. It has been repeatedly observed (*Heilbronner*, *Hans Curschmann*), that in the later generations the descendants are attacked at an earlier and earlier age. On the other hand, members of choreic families display neuropathic tendencies, suffer from hysteria, dementia, paranoia.

Age and Sex.—In contrast to infectious chorea, hereditary chorea begins at a *more advanced* age, usually between the 30th and 45th years. The *male* sex seems to be somewhat more frequently attacked.

Symptomatology.—The center ground of the disease picture is occupied by the muscular twitchings, which, in their character, closely resemble those of infectious chorea. They are involuntary, inco-ordinated, almost uninterrupted persistent movements of the most varied muscular groups. On the whole, they seem to be somewhat slower and more sluggish than in infectious chorea. The twitchings affect especially the muscles of the *face* and the *extremities*. The choreic twitchings of the forehead and mouth, the wide opening of the eyes and the smacking noises made with the tongue and lips lead to peculiar *grimaces and gesticulations*. As a result of the choreic restlessness of the tongue, throat, and the respiratory muscles, speech is made difficult, the sentences are chopped off, the words thrust forth in syllables; in writing the lines are crooked. The patients walk with legs wide apart, tottering or tripping; now they move slowly forwards, now they jump forward now they suddenly cease. This peculiar walk with the dangling arms and grimaces may remind us of the behavior of clowns.

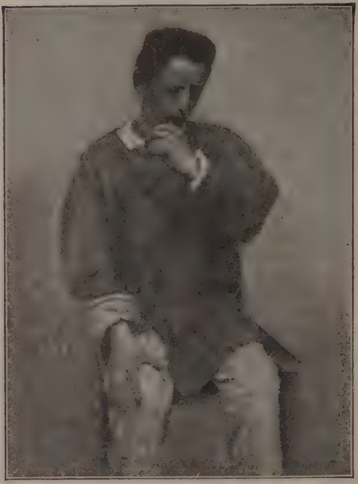

FIG. 207.—Chorea Huntington depressive form with less theatric than "embarrassed movements."

The choreic twitchings disappear in sleep; excitement increases them, but *in intentional movements* the patients are able in part to lessen the motor irritation phenomena, in part to wholly suppress them. Thus it happens, that for a comparatively long time the patients are able to perform certain complicated movements, and to follow certain vocations. The coarse muscular strength and electric excitability of the nerves and muscles remain intact; in a few cases, there appear towards the end of life, paralyses mostly of a hemiplegic nature. The tendon reflexes are usually somewhat increased. The internal organs, bladder and bowels remain normal; there are no pathological changes in the heart.

Almost invariably, *psychic disturbances* accompany the choreic twitchings. The patients become apathetic, depressed, sometimes even extraordinarily irritable. Later, there appear loss of memory and psychic weakness; often also there is such loathing of life as leads to an attempt at suicide. In the course of years the patients usually reach the stage of *mental weakness and idiocy,* in which delusions of persecution and megalo-mania may clearly appear. The patients then sit absolutely apathetic; they must often be fed artificially; the choreic twitchings, on the other hand, are frequently diminished.

Course.—The disease exhibits a chronic progressive course, that may extend over 20 years and more, with an absolutely *unfavorable prognosis.* Some patients die of intercurrent diseases and traumatism, others of general cachexia. The latter is often increased by insufficient nutrition, which is a result of the choreic restlessness of muscles subserving deglutition.

Diagnosis.—The proof of heredity, which can generally be found, the slow and progressive development of the disease, and the gradual appearance of a disturbance of the intellect, all speak for hereditary degenerative chorea. If these distinguishing features are missing, it is occasionally difficult to differentiate between *infectious* and hereditary chorea, since, though it rarely happens, cases of infectious chorea may become chronic. Here, if we assume an hereditary chorea, we shall be supported by the gradual aggravation of the disease, the severe psychic alteration, and, to a certain degree, by the slower character of the twitchings. Exceptionally, there is in chorea hereditaria only slight disturbance of the intellect.

Pathological Anatomy.—Various anatomic changes have been found in the nervous system, the connection of which with the phenomena of the disease is not yet clear. Among such changes are: cloudiness of the meninges, adhesions of the same with the cranium and the brain, hemorrhagic pachymeningitis, hydrocephalus externus, *disseminated encephalitic foci,* becoming transformed into sclerosis of the central convolutions (*Oppenheim, and Hoppe Kronthal and Kalischer, Facklam*), as well as the phenomena of *diffuse encephalitis.*

Treatment.—The treatment is purely *symptomatic.* Occasionally, *arsenic* and *hyoscin* (subcutaneous injection of 0.0005–0.002 pro die) may have a favorable effect upon the process. Besides this, *medical gymnastic* exercises are recommended. In case conditions of excitement appear, luke-warm *baths, bromides and opium* are in place. In conditions of anxiety, and clearly appearing psychic disturbances, the placing of the patient in a suitable *institution* may be necessary, since there is great danger of *attempts at suicide.*

(c) Chorea Electrica

Various diseases of unknown origin have been described under this name. *Henoch* separated from the ordinary form of infantile chorea a disease picture which he called chorea electrica, in which the muscular twitchings in contradistinction to those in infectious chorea follow one another with lightning rapidity; they affect preferably the muscles of the shoulder and the neck. Most probably, myoclonia is the disease concerned in these cases. *Bergeron* described as chorea electrica a disease, that attacks delicate, anæmic children. The sudden, jerky twitchings in the muscles of the neck, the shoulders and the arms, shake the entire body; occasionally however only one extremity is affected; the intention to overcome them not only fails, but usually even aggravates the phenomena of the disease. Arsenic and hydrotherapy sometimes overcome the trouble after some time. Presumably hysteric chorea is present.

Finally, *from upper Italy*, a disease picture has been reported (*Dubini*), beginning with pains in the region of the back and the neck, which leads to lightning-like spontaneous twitchings, that start in one-half of the body, going from above downwards; usually spreading from there to the face, the arm and the leg on the other side. Occasionally, also epileptiform attacks and paralytic phenomena are added. The disease, which is characterized by fever, leads in a shorter or longer period of time to death; recovery is rare. It is probably a question of an infectious disease of unknown origin.

2. TETANY

Tetany manifests itself by *tonic muscular spasms*, which appear paroxysmally and affect especially the *arms*, while consciousness usually remains intact.

Tetany occurs in *certain vocations and conditions*, and accordingly the following forms are distinguished clinically: *artisan's tetany*, *gastro-intestinal tetany*, tetany in *acute infectious diseases*, *tetany of pregnancy*, tetany after *intoxications*, *infantile tetany*, and operative tetany after *extirpation of goiter*.

Artisan's tetany attacks otherwise healthy, mostly youthful persons of the working population, especially those engaged in certain occupations such as shoemaking (hence the name "*shoemakers' spasm*"), tailoring, carpentering, turning and locksmithing; it *appears epidemically* during certain months (*January to April*) and prefers *certain places* in central Europe, especially Vienna and Heidelberg. Tetany in *gastro-intestinal diseases* occurs in disturbances of the bowels (diarrhœa, and constipation), and especially in stomach ectasias after stenosis of the pylorus (as a result of ulcer or carcinoma) (*Kussmaul*). Tetany that appears in the train of *acute infectious diseases*, is comparatively rare and is observed in typhoid, cholera, measles, influenza. *Tetany of pregnancy* is comparatively common, developing during pregnancy or lactation, inclines to recurrence, and like artisan's tetany, appears with special frequency in the months between

January and April, and also shows a preference for some places as, for instance, Vienna and Heidelberg. Tetany after *extirpation of goiter* (*N. Weiss*) (Tetania strumipriva) is to be traced back, as the investigations of the last decade teach, to removal or lesion of the *parathyroid glands* (*glandulæ parathyreoideæ*) (*tetania parathyreopriva*). *Infantile tetany* usually attacks rachitic infants suffering from intestinal troubles, and is often accompanied by spasm of the glottis and convulsions.

Symptomatology.—Tetanic muscular spasms are frequently ushered in by *prodromal phenomena*, which manifest themselves as painful sensations and paræsthesias in the realm of the diseased member. *Hands and arms* are preferably attacked by the spasm, especially the musculi interossei and the other small muscles of the hand. There is flexion of the proximal phalanges, extension of the middle and distal phalanges, as well as adduction and opposition of the thumb, from which arises that characteristic position, which is known as the *accoucheur's hand or paw-position.* Often the legs also are attacked, and then especially the flexors of the feet and the toes. In the higher degrees, other muscular groups, such as the *muscles of the face, the eyes, the trunk, the throat, and respiration,* are also involved in the spasm: spasms of the ciliary muscles occur also, and cause characteristic visual disturbances. Less common are *choking, laryngeal and intention spasms* (*F. Schultze, J. Hoffmann*). The latter are characterized by the fact, that tonic spasms are evoked through intentional movements, disappearing only after some time. The *symmetrical* appearance of tetanic muscular spasms is striking; it very rarely affects only one-half of the body. If the spasm is but of slight intensity, it is easy to master the muscular tension; but if the attacks are more severe, the usually painful spasm can not be made to relax even with the greatest exertion. The duration of the spasm fluctuates between minutes, hours, days. Afterwards there often remains an annoying feeling of tension or formication. In adults, there are now and again *epileptiform* attacks, during which consciousness is impaired; on the other hand, eclamptic attacks are far more common in children, who suffer from tetany.

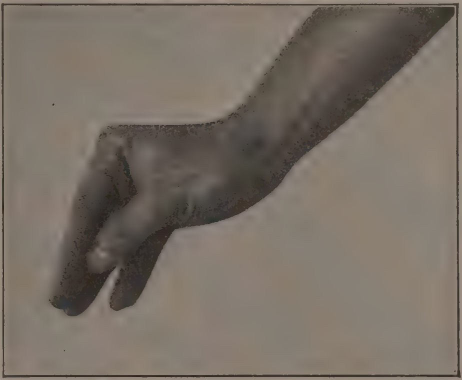

FIG. 208.—Hand in tetany contracture. (Medical clinic, Heidelberg.)

Slight *increases in temperature* are observed rather frequently; subnormal temperatures also occur occasionally.

Besides the spasms, which are the most striking symptom of tetany, there are still many characteristic symptoms, which accompany the muscular spasm, but may exist alone (*latent tetany*), above all, Trousseau's, Erb's, and Chvostek's phenomenon.

Trousseau's sign is brought out by pressing the nerves in the sulcus bicipitalis with the fingers, or, still better, by tying up the arm of the patient by means of an Esmarch bandage or a rubber tube; after some seconds or minutes, a typical spasmodic attack will be excited in the arms and hands in most cases. It is most probably (*v. Frankl-Hochwart*) the pressure upon the nerves and not the compression of the vessels, that induces the attack. Trousseau's phenomenon is pathognomonic for tetany, as it has never been elicited in any other disease, but it may occasionally be lacking in tetany, even though there is no doubt as to the diagnosis.

Erb's phenomenon consists of an *electric overexcitability* of the motor nerves, which is manifested by the fact, that the K. C. C. appears even upon the application of a weak current, and very easily passes over into K. C. T.; likewise, A. O. C. and A. C. C. appear in relatively weak galvanic currents. Thus, for instance, the ulnar nerve of the healthy individual reacts to K. C. with 0. 9–3, 3 milliampères, whereas in tetany patients, K. C. C. is obtained even at values of 0. 1–0. 7 milliampères. In children (*Mann*, *Thiemich*) the appearance of K. O. C. below 5 milliampères and the prevalence of A. O. C. over A. C. C. is characteristic of tetany; similar conditions are occasionally found in tetany of adults also (Pineles).

Chvostek's phenomenon consists of the *mechanical overexcitability* of the nerves, which is especially clear in the facial nerve (facialis phenomenon). If with a percussion hammer, the facial trunk is tapped in front of the external auditory meatus, one observes in the highest degrees of overexcitability, twitchings in the entire facial area (Chvostek Nr. 1)[1]; in some cases, the phenomenon may be evoked by gently stroking the face with the hand. In medium degrees, there are only twitchings in the alæ nasi and the corners of the mouth (Chvostek Nr. 2), while in a slight degree of overexcitability only twitchings of the corner of the mouth (Chvostek Nr. 3) are demonstrable. Chvostek's sign is occasionally found in individuals, who show no other sign of tetany, for instance in enteroptosis (*Mager*).

An increase in the mechanical and electrical excitability of the *sensory nerves* is also frequently present (*Hoffmann's symptom*). Pressure on sensory nerves then evokes paræsthesias and painful sensations in the regions, that are supplied by the nerves in question. Occasionally, the faradic and galvanic excitability of the *nerves of special sensation* (that is, the auditory) is increased (*Chvostek Jun.*).

There are also *secretory and trophic disturbances*. Thus, both during the attack and in the free intervals, are often noted outbursts of perspiration, edema of the eyelids, urticaria, swelling and cyanotic discoloration of the hands. Very characteristic is the peculiar *bloated appearance* and swelling of the face, which is observed with particular frequency in artisan's tetany ("*tetany face*"). Occasionally in sufferers from tetany there appear changes in the *hair and nails;* the hair becomes thin; the growth of hair decreases and under certain conditions the hair entirely disappears. The nails become thin, brittle, and fall out. Both, nails and hair, however, grow again, frequently disappearing again upon a recurrence of the disease. In some cases, *cataract* is formed (*tetany cataract*). It is characteristic of these trophic disturbances that they appear almost exclusively in chronic recurrent tetany and in all forms of tetany, even in parathyreoprival tetany.

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[1] As suggested by *Frankl-Hochwart*.

Only very rarely are paralyses and atrophies observed in individual muscles (of the buttocks and abdomen); neuritis optica and mydriasis are also rare findings. The *tendon reflexes* are at times increased in the acute attacks, but may also be diminished or absent. Cutaneous and mucous membrane reflexes are normal. The *psychoses* that appear here and there in the train of tetanic muscular spasms, run their course under the picture of confusion with hallucinations, and are to be reckoned among the toxic deliria (*v. Frankl-Hochwart*).

Course and Prognosis.—In artisan's and pregnancy tetany the prognosis in some cases is very *favorable*, since the disease, after a longer or a shorter time is cured. But in all the rest of the cases observed—no inconsiderable number—the course is *chronic, recurrent*, and in the course of many years, the disease appears again and again, usually at the above-mentioned tetany season (*v. Frankl-Hochwart*). Even in the intervals, that are free from spasms, there are present in many patients, symptoms of the disease: paræsthesias, painful feelings of tension, attacks of profuse perspiration, general weariness. In this form, trophic changes are comparatively frequent (cataract, loss of hair and nails). On the other hand, the prognosis quoad vitam is favorable in so far, as no fatal case of idiopathic tetany without other complications has been hitherto observed. The tetany in *diseases of the stomach* has a far less favorable prognosis, since the gastric disease in itself represents a severe often fatal disease (pyloric-stenosis, carcinoma ventriculi). In *operative* tetany, after extirpations of goiter, the prognosis depends upon the amount of the remaining parathyroidal tissue, that is still functioning, wherefore total strumectomy, which has now been abandoned, had far worse results than the partial method now in use, in which an attempt is made to spare the parathyroids as much as possible. In *infantile* tetany, the eclamptic attacks, occasionally too, the glossal spasms bring about death.

Etiology and Pathogenesis.—The fact, that the appearance of tetany is preferably restricted to certain places and to a certain time of the year (January to April) (*v. Frankl-Hochwart*), as well as the fact that tetany is occasionally observed in members of the same family, or of the same district, furthermore, that some years are unfavorable, others favorable for its occurrence, finally that it prefers members of certain vocations, all these things speak emphatically for the assumption of an *infectious element* in the production of the disease. Whether this infectious virus is connected with the small and badly ventilated houses used in the vocations chosen by the victims of tetany, whether it should be sought in the materials they work in (*Oppenheim*), or whether other ætiological factors are concerned, has not yet been established.

In respect to the pathogenesis of the disease, the assumption of a common pathogenetic foundation for all forms of tetany is justified. In considering this theory, it must be recalled that between the various clinical forms and the parathyreoprival form of tetany, there is striking uniformity, so far as many of their symptoms are concerned (hands in accoucheur's position, Erb's, Chvostek's, and Trousseau's phenomena, intention spasms, cataract, falling out of hair and nails, etc.). Since parathyreoprival tetany may be traced back to the loss of the *parathyroid glands*, we must assume intimate *pathogenetic* relations with these glands in idiopathic tetany also.

The *parathyroid glands* (glandulæ parathyreoideæ) are small glandular organs, which lie partly near the thyroid glands, partly in them. The "external" parathyroid glands, lying outside the thyroid gland parenchyma, were dicovered by the Swedish anatomist *Sandström* (1880), and the first physiological investigations (1892) by *Gley* showed that these organs have a very important specific function. But only the significant discovery of the "internal" parathyroid glands by *Alfred Kohn* in Prague (1895) made possible the execution and the correct interpretation of the experiments of *Moussu* (1896–98) and *Vassale and Generali* (1896–97), which connected the symptoms of experimental tetany with the loss of the parathyroid glands. These results were confirmed by numerous investigators (among others, by *Welsh*, *Biedl*, *Pineles*, *Erdheim*). *Pineles* transferred the results of experimental investigation to human pathology (1904) and showed—his proof involved experiences in the disease picture of thyreoplasia and partial strumectomies, as well as the observation of tongue goiters—that tetany strumipriva must be traced back to the loss of the parathyroid glands. *Erdheim* (1906) confirmed this by anatomic examination of cases, in which, in connection with strumectomy lethal tetany had developed. Furthermore, *Pineles* (1904–06) proved, that there is such a striking uniformity between the experimental tetany of animals, the human strumiprival (parathyreoprival) tetany and the various forms of human idiopathic tetany, not only in respect to the nervous symptoms (phenomena of Erb, Chvostek, and Trousseau, isolated and general muscular spasms, position of the hand known as accoucheur's hand, chronic spasms, pareses, myotonic reactions, epileptiform attacks), but even in respect to the trophic disturbances (formation of cataract, falling out of hair and nails), that the "parathyreoprival tetany poison" (i. e., that poison, which develops its injurious effect upon the organism after the loss of the parathyroid glands) must be regarded as the general physiopathological substratum of the various human tetanies. To this belief, Erdheim's investigations (experimental formation of cataract, loss of hair, appearance of pregnancy, tetany in rats, that have lost their parathyroid glands) have given further confirmation.

The hypotheses offered as an explanation of gastric tetany (reflex theory, theory of the thickening of the blood) have proved to be untenable.

Pathological Anatomy.—The examination of the nervous system has not yet produced any results worth mentioning. Changes have been found in the ganglion cells in the spinal cord, hemorrhages, softening foci, etc. The microscopic findings in the parathyroid glands have been to an extent negative; in infantile tetany, the presence of hemorrhages has been frequently demonstrated (*Erdheim*, *Yanasse*).

Diagnosis.—In by far the greater number of cases, the diagnosis of tetany can be easily made. Paræsthesias and drawing pains, the symmetrical spasms in the arms, the paw position of the hands, the intactness of the sensorium, and finally Erb's, Chvostek's and Trousseau's phenomena are the most important points of vantage for diagnosis. Difficulties arise sometimes in respect to the differentiation of tetany from *hysteria*, *epilepsy and tetanus*. There are definitely established mixed forms of *hysteria* and tetany, in which, besides the symptoms characteristic for tetany, signs of hysteria are present (as for instance general convulsions with opisthotonos, hemianæsthesia, hysterogenic zones). There are, furthermore, cases of hysteria that simulate tetany. The following are symptoms in favor of hysteria: the immediate, instead of gradual appearance of Trousseau's sign after the application of the compression bandage, which frequently leads not to the characteristic paw position, but to a spasmodic fist formation, the absence of paræsthesias, that accompany Trousseau's phenomenon, the possibility of evoking the phenomenon in other parts of the body (for instance, through pressure on the region of the ovaries), the absence of Erb's and Chvostek's phenomena. The differentiation of severe tetany from the *epileptic* attack will be difficult only in very rare cases. In the epileptic attack, there is instantaneous total loss of consciousness, frequently injuries, bite of the tongue, incontinence of the bladder and the bowels, and postepileptic confusion. On the other hand, the epileptiform attack in tetany is ushered in with paræsthesias in the arms, shows a gradual dimming of the sensorium, and typical accoucheur's position of the hand, while involuntary discharge of urine and feces, injuries of the tongue, and conditions of confusion are absent. In very severe tetanic muscular spasms the differential diagnosis between tetany and *tetanus* might also come into play. Tetanus shows a peculiarly severe involvement of the muscles of the jaw and back of the neck, and extensor spasm in the fingers, whereas the phenomena of Erb, Chvostek, and Trousseau are missing.

Finally those cases of tetany must be referred to, which are designated as "*formes frustes*" or "*tetanoid*" conditions and the diagnosis of which may be difficult. It is a matter of latent, slight degrees of tetany, in which there are no real spasms, and only the history of the patient in respect to paræsthesias, together with the proof of the presence of Chvostek's and Erb's phenomena assure the diagnosis of tetany. Probably those individuals who are found in great numbers in cities noted for tetany, and present only the isolated Chvostek's phenomenon, belong to this group.

Treatment.—In all forms of tetany, the administration of bromides in not too small doses for lessening the severity of the attacks, is recommended (natr. brom. 3.0–5.0 pro die); in severe cases, *morphine, hyoscine, or curarine* (0.3–0.6) may also be tried. Besides, *warm or hot baths* are recommended, *hot packs*, and lacto-vegetable diet, with avoidance of meat-broths and meat (parathyreoprival persons have a distaste for meat, and parathyreoprival animals show a decrease of spasms with a diet, that is devoid of meat). The use of coffee, tea, and alcoholic drinks must be forbidden. Treatment with *thyroid gland tablets* (1–2 tablets daily) and with *parathyroidal preparations* (parathyreoidin tablets of Vassale, 3 times a day 2–3 tablets, parathyreoidin solution of Vassale, 3 times daily 20–30 gtts.) has proved to be useless. For opposing the occasionally very severe pain, *salicyl, phenacetin, and antipyrin* suffice in light cases; in serious cases, *morphine* must be used.

In artisan's tetany, hygienic measures must also be considered in respect to *prophylaxis*. One should see to it, that the people do not live crowded together, and that there is thorough ventilation of the living and working rooms; also that the patients do not expose themselves to the influence of cold, and avoid the use of alcoholic drinks. In cases that incline to recurrence, a change of residence and vocation must be insisted upon. Mothers, who suffer from *lactation tetany*, are forbidden to suckle their babies, and are recommended, especially in the recurrent forms, to avoid another pregnancy. The internal administration of *phosphorus* is earnestly indicated with children, who suffer from tetany.

In *gastric tetany*, the careful employment of soft stomach tubes and washing out the stomach with a 3% solution of boric acid, or a 1% solution of thymol are recommended; but it must be remembered that the introduction of the tube frequently leads to the excitation of tetanic spasms. Subcutaneous injections of sterilized *physiological salt solution* are very serviceable as well as *rectal injections of milk and water*, since they are able to replace the masses of fluid removed from the stomach. If the condition does not improve, *operative* interference (gastroenteroanastomosis, resection of the pylorus) must be considered, which, as a matter of fact, in some cases, succeeded in removing the tetany.

Parathyeoprival tetany is prevented by avoiding as far as possible removal or lesion of the parathyroid glands. It is necessary therefore to seek the avoidance of the removal of the parathyroid glands and to spare the N. recurrens. If, in spite of this care, there is an outbreak of tetanic spasms, the *implantation of human parathyroid glands*, that were accidentally removed in extirpations of goiter, is urgently indicated (*v. Eiselsberg*, *Garré*). In chronic idiopathic tetany also, the same curative means should be considered.

3. PARALYSIS AGITANS (SHAKING PARALYSIS)

Paralysis agitans (also called Parkinson's disease) is a rather rare disease, which appears usually in the presenile years (between the 45th and 60th years); there are but very few cases below the 40th year. It attacks the male sex somewhat more frequently, than the female and by preference the poorer classes of the population.

In respect to **etiology,** definite essential facts are yet lacking. Sometimes the disease is supposed to have developed in connection with intense psychic excitement (fright, grief, a life of worry), or great physical over-exertion (v. *Krafft-Ebing*). In other cases, physical traumatism and severe chilling are accused as causes of the disease; occasionally too there is supposed to be a connection with infectious diseases. Hereditary relations do not play any considerable rôle. From the variety of the ætiological causes cited, it is evident that, at best, they represent only exciting factors.

Symptomatology.—Occasionally, the disease is preceded by *prodromal symptoms*, which are manifested by sensations of pain in the most varied places, paræsthesias and severe fatigue. Usually, the disease phenomena appear very gradually, beginning with trembling in the hand and the fingers of one side. At the beginning the *trembling* is generally slight, may even cease for a time, but gradually it increases in intensity, and spreads slowly to other groups of muscles. Soon a *rigidity* appears in the trembling muscles, which is accompanied by a slowing of movements. After a shorter or a longer time, the other limbs begin to tremble also, and finally a large part of the muscles of the body is thus attacked. If the disease is even half-way developed, the patient presents a rather striking picture.

He stands rigid, statuesque, as if lifeless. The *face* is motionless without any play in the muscles, as if covered by a waxen mask, the mouth closed, and only the eyes behind these mask-like features look so bright, as to betray the intactness of intellect. In the hemiplegic form, the rigidity may be confined to one half of the face. The rigidity of the features is interrupted by trembling movements of the lower jaw, tongue and lips. *The posture of the body* is stooping, the trembling head and trunk are bowed forwards, the arms pressed to the trunk, somewhat flexed at the elbows, the hands in extension, the fingers in paw position (metacarpo-phalangeal joints flexed, middle and end phalanges in extension); hands are affected by violent trembling movements, in which the fingers are flexed and extended, welded together, or spread apart, index fingers and thumbs meet in a revolving movement, as if they were kneading something. Legs, somewhat flexed at the hip and the knee, are also attacked by the characteristic trembling, though less severely than the hands; the thighs are adducted. The movements of the patients are slow and dragging, and a turning of the body is effected by several tripping steps. The gait is slow, as if upon rigid or anesthetic soles, which deprives it of its certainty.

These disease phenomena gradually become aggravated, the rigidity of the muscles increases, the patients finally execute even slight movements only with great trouble, become incapable of changing their position without assistance, become bedridden and have to be fed and otherwise attended to, and suffer from sleeplessness. Death results from marasmus, or from intercurrent diseases.

To the most important disease symptoms belong: trembling, stiffness in the muscles, as well as the retardation and impairment of movements.

Tremor, the most striking initial symptom, is absent only in extraordinary cases (paralysis agitans sine agitatione). It shows a number of characteristic peculiarities. First of all, the oscillations are slow, occurring about 4–5 times a second. They are characterized by rhythm and regularity, thus giving the trembling a stereotyped appearance. The tremor is constant during both rest and movement, interrupted if at all but for a very short time, and without any apparent cause. But at least it can be seen, that active movements occasionally lessen the tremor, or may even check it altogether; thus the firm intention to perform a movement may have a favorable inhibitive influence upon the tremor, with the exception of the more complicated movements as in writing. In the same way, tremor may be controlled by passive movements, but often simultaneously give rise to an increase in the limbs, that are at rest. Every sort of psychic excitement invariably has a tendency to increase the trembling, sleep nearly always causes it to stop.

If tremor is the most striking and usually the first symptom, *muscular tension* is the most constant and to this extent the most important phenomenon, in that in the rare cases in which tremor is missing, it is the only characteristic sign of the disease and in that it is the cause of the above described facial expression and position of the body. This muscular stiffness attacks a large part of the musculature, especially the muscles of the extremities, face, neck and spinal column. Partly dependent upon the muscular stiffness, partly, however, independent of it is *awkwardness* of the active movements, which scarcely permits the patient to obey the impulse of the will, and is most noticeable, where complicated manipulations are concerned. While examining the patient for muscular stiffness through passive movements of the limbs, the investigator notices that the rigor has the peculiarity, that the resistance of the limbs does not change according to whether they are moved slowly or rapidly, but it remains constant; this might indicate, that we should not look for the ultimate cause of the symptom in the nervous system, as we do in other spasms, which increase upon passive movements and, in fact, which are often only evoked by them. Another difference also between the two kinds of spasms would speak for this supposition, namely, that the spastic rigor in diseases of the nervous system (hemiplegia, paraplegia) is accompanied by an increase in the tendon reflexes, while in paralysis agitans, the tendon reflexes are mostly unchanged.

In the pronunced, severe cases of paralysis agitans, the *gait* of the patient frequently shows the following disturbances: the patient, asked to walk across the room, begins with slow, thoughtful steps, hurries more and more, and finally he would pitch forwards, if he did not grasp some support (*propulsion*). The same type of movement occurs on going backwards (*retropulsion*), less often laterally (*lateropulsion*). Each of these different forms may also be evoked, by giving the patient an initial push in the desired direction. Probably these phenomena are to be traced to two different causes, one of which lies in the patient's being constrained by the muscular tension, which second depends upon the fact, that the impulse of the will produces motion in a far more difficult and troublesome way than in healthy persons.

Speech often shows some deviation from the normal. The *voice* is weak, tearful, monotonous. The patient is able to speak only some time after the thought has been created; once he begins to speak, the words fairly tumble over each other.

The *tendon reflexes* are always preserved, sometimes increased. An actual ankle clonus can never be evoked, through it ought to be mentioned, that here and there the investigation of this symptom leads to the characteristic trembling movements of the muscles of the feet ("*false foot tremor*," *Oppenheim*). Frequently the *paradoxical foot phenomenon* is demonstrable, in that the foot, when dorsally flexed remains for a long time in its position as a result of tonic contraction of the extensors of the foot. The Babinski phenomenon is always absent. The galvanic and faradic muscle contractity remains normal.

Disturbances in the *function of the bladder* occur occasionally, but are always to be traced to complications (for instance hypertrophy of the prostate). *Sensibility* has been found to be altered in a few cases (*Ordenstein*, *Karplus*) (hypæsthesia and hypalgesia in the extremities). In the realm of the *sense organs*, disturbances due to the disease do not occur (for instance atrophy of the optic nerve or nystagmus).

The patients frequently complain about a *feeling* of excessive *heat*

(*Charcot*) and show an increase in the *secretion of perspiration;* sometimes increases in temperature have been observed.

The *intellect* is not impaired; it is natural enough, that the mood of the patient should be very gloomy owing to the annoying troubles. *Psychic disturbances* do not belong to the disease picture of paralysis agitans, but are due to complications.

Course.—Paralysis agitans is a very *chronic*, *progressive* disease; the prognosis quoad vitam is good, quoad sanationem unfavorable, since recovery never occurs. Occasionally remissions are observed, which are, however, soon followed by an aggravation of the disease. Mostly many years (usually 15 to 20) pass, before the patient as a result of the high degree of muscular stiffness becomes bed-ridden. Apoplectiform attacks occasionally appear. If they lead to hemiplegic phenomena, the trembling may, for a time, cease here and there in the hemiplegic limbs, only to appear again.

Pathological Anatomy.—Nothing certain has as yet been discovered as to the anatomic foundation of the disease. In some cases (*Koller*, *Redlich*, *Lauder and others*) *sclerotic processes in the vessels* and proliferations in the glia and connective tissue, especially of the spinal cord, were found to exist; these changes, however, correspond to the disturbances in the nervous system that appear in senility. Therefore, according to some, the condition may be recognized rather as a higher degree of senile degeneration (*Dubief*, *Borgherini*, *Ketscher*). On the other hand (*Gauthier*, *Blocq*, *v. Sass*), attempts have been made to connect anatomic changes in the muscles with the disease picure. Some authors (*Lundborg*, *Möbius*) have called attention to the *thyroid and parathyroid glands* in connection with this disease.

Diagnosis.—In the establishment of the diagnosis, there are sometimes difficulties. Thus, *senile tremor* may present a picture that resembles paralysis agitans; it should be remembered that in it the trembling affects the head mainly, is increased by intentional movements, very rarely appears on one side only, and that the muscular stiffness is missing. Furthermore, *senile atherosclerosis of the nervous system*, especially in respect to the posture of the body, occasionally reminds the physician of paralysis agitans. But in it, there are always some paralyses, such as disturbances in deglutition, dysarthria, bladder disturbances, to be found. *Progressive paralysis* also at times exhibits trembling movements, which, however, have no rhythm or regularity, while, on the other hand, the changes in the psyche and the characteristic paralytic speech disturbance are lacking in paralysis agitans. The tremor of *multiple sclerosis* subsides during rest, and, in contrast to that of paralysis agitans, appears during intended movements; the characteristic symptoms of multiple sclerosis, such as nystagmus, optic atrophy, weakness of the bladder, are never demonstrable in shaking paralysis. *Hysteric trembling* is differentiated from the trembling of paralysis agitans, in that the oscillations are much stronger, are intimately connected with psychic processes, and may be overcome by hypnosis. Finally, it should be mentioned, that propulsion and retropulsion occasionally, even though in a slighter degree, are found in *neuroses* and in diseases of the *cerebellum.*

In **treatment** one should see to it especially, that the patient gets physical and mental rest. Psychic excitement and noisy surroundings are to be avoided. An *isolated* life in the country is best, perhaps in forest covered mountainous regions—in the early stages, moderate movement in the fresh air. Moderate *cold-water treatment* (luke-warm hip baths with somewhat cooler showers), *electric baths*, careful *passive movements* with frequent variations (*Oppenheim*), light *massage* and vibratory treatment influence the condition favorably. Since people who suffer from shaking paralysis are supposed to endure riding in carriages and trains exceptionally well (*Charcot*), the use of especially constructed chairs (fauteuils trepidants), which by continuous oscillations are believed to ease the patient, is recommended by many. Suspension and nerve stretching are to be rejected under all circumstances (*Oppenheim*).

As to *medication*, *bromides* are used to quiet the patient and to allay the conditions of anxiety. In some cases, the trembling is *favorably* influenced by: *tinctura veratri* (3–6 gtts. several times daily), *tinctura Gelsemii*, *sol. arsen. Fowleri* (sol. ars. F., aq. fœniculi, tct. nuc. vomic. āā 5.0 daily 6–12 drops), *hyoscin* (scopolamin, hydrobromic) three times daily 0.3 mg internally or subcutaneously, *Duboisin* sulf., 3 times daily, 0.3–0.4 mg in pills or subcutaneously, *rhizoma scopoliæ carniol* (three times daily 0.2–0.4).

With the last three drugs, one should watch for the appearance of toxic symptoms (like headaches, dizziness, nausea, visual disturbances). Feeding the substance of the thyroid and the parathyroid glands has not benefitted the patients. In severe cases, in which the trembling movements influence the condition unfavorably, and disturb the rest at night, hypodermic *injections of morphine* must be resorted to.

4. TREMOR

(a) Habitual Tremor

In young persons, there is occasionally found a tremor, which attacks preferably *hands and head*, but less frequently also the *muscles of the face and the tongue.* It has shallow oscillations, subsides at rest, and is increased by voluntary movements and psychic excitements. Occasionally the patient is able to lessen it by the exercise of his will. The tremor after persisting for some years, may disappear forever, or after a long remission, appear anew; often it persists throughout life.

Nothing definite is known as to the ætiology of this tremor. It is found mostly in *neuropathically* predisposed individuals, who sometimes trace their illness back to great excitements (sorrow, fright). Bromides and arsenic are supposed to influence the disease favorably.

(b) Familial Tremor

Familial tremor is a rare disease, as to the causes of which we are still completely in the dark. Occasionally there are present in some of the ancestors chronic alcoholism, psychoses and epilepsy. Both sexes are predisposed in *like* degree; likewise, the heredity passes down through the male and female sex alike.

The tremor usually appears in youth, less frequently does it begin at a more advanced age. It is characterized by rhythmic oscillations extending over a small space, and appearing from three to nine times a second. In a condition of rest, the tremor sometimes diminishes; it may frequently be suppressed by the will of the patient, but almost regularly becomes augmented in intended movements. In some cases, an *intention tremor* is present, in which the trembling is absent at rest, and is noticeable only during the execution of movements.

The *hands* are most frequently and most severely attacked, the *legs* also with relative frequency, while the muscles of the *face* and the *tongue* are more rarely involved in the trembling movements. It happens occasionally, that the trembling movements are combined with tic-like and choreic twitchings.

The disease is transferred from generation to generation, and is present often in several members of the same generation.

Sometimes the tremor persists throughout life and may show a slowly progressive course; in other cases, a slow, lasting improvement appears, which occasionally leads to total disappearance of the tremor. Treatment seems to be absolutely powerless.

(c) Senile Tremor

Old persons occasionally present a tremor, which affects especially the head and the arms. It is a tremor with *fine oscillations*, which diminishes or entirely disappears during rest, and is increased by voluntary movements. During sleep, it is absent. It can not be influenced by medicinal or other therapeutic measures. It is distinguished from the tremor in paralysis agitans, in that rigidity and weakness of the muscles are lacking.

The various forms of tremor, which occur in organic affections of the central nervous system, in hysteria, and in chronic intoxications (alcohol, lead) are discussed in the chapters devoted to these diseases.

5. OCCUPATION CRAMPS (OCCUPATION NEUROSES)

To the nervous disturbances, which seem most likely to be evoked by accidental influences, and which, less than any others, seem to form a part of general nervous affections, belong occupation cramps. Still the fact can easily be demonstrated that they always attack individuals, who, in one way or another, are nervously predisposed, for which reason often more than one member in a family is attacked.

Occupation cramps are characterized by cramp-like conditions in the muscles, which appear only in *quite definite*, complicated, muscular movements, that have been *learned by habit*, whereas all other actions performed with the same muscles are perfectly normal. Therefore the name suggested by *Benedikt* "*co-ordinated occupation neuroses*" seems very fitting. To this group belong the occupation cramps of *telegraphers*, *seamstresses*, *tailors*, *shoemakers*, *milkers*, *cigar wrappers*, *dancers*, *etc.* In tailors and seamstresses, the innervation disturbances affect the muscles used most, those of the thumb and the forefinger; in milkers, there are paræsthesias in addition to cramps in the muscles of the hand and the fingers. *Pianists' and violinists' cramp* are still more common, while *writers' cramp* (*graphospasm*) is the most important and striking. In this condition it is a cramp-like state, which interferes with writing, since it affects those muscles of the hand and fingers that are used in writing. There are concerned, in the first place, the musculi interossei, and lumbricales, the thenar and antithenar, the flexors and extensors of the fingers and the wrist, as well as their pronators and supinators. The script is distorted by the cramp, though other movements are left undisturbed. The main rôle as exciting factor is played by the vocational overirritation of the groups of muscles concerned, but also accidental factors such as unsuitable, or too inflexible a pen, too energetic a pressure upon the pen, or even upon the fingers not employed in writing (third and fourth fingers) are often to be blamed. It is very reasonable to suppose that excitement may contribute to the origin of the trouble.

In most cases, the patient feels first only *fatigue* in writing, a lack of control over his writing, which can not be performed as was formerly the case and there develops gradually a more and more cramp-like muscular tension of the flexors of the fingers (especially of the thumb and forefinger), without any relief to the patient. Now and then, on the other hand, the penholder falls from the hand, because the fingers become similarly cramped in a position of extension. Later, usually, the *wrist* and the *forearm* are also affected and *pains* appear, sometimes also *weakness or tremor* in the hand. Accordingly, one differentiates *spastic*, *tremor-like*, *and paralytic* forms of graphospasm.

The *objective findings* show scarcely anything at all; therefore, the disease must be regarded as an *exhaustion neurosis*, to which no anatomic foundation corresponds. This disease, moreover, may last during a lifetime, without any other disturbances in health. Sometimes the disturbance is alleviated for a time, if the left hand becomes accustomed to do the writing, but later the cramp generally spreads to it also. Therefore, as only a few cases recover, and even these are not protected against a recurrence, a favorable prognosis can usually not be given.

Diagnosis.—Of course many things resemble writers' cramp, that owe their origin to quite different diseases (such as tabes, multiple sclerosis, paralysis agitans) or even functional tremor may simulate it, in which, however, the trembling is not confined to writing movements, and which may be influenced by suggestion.

Treatment.—First of all, writing must be forbidden for a long time, and, since many individuals attacked by writers' cramp have a general nervous predisposition, it is advisable to oppose the neurosis by *hydriatic* procedures and *tonic* medicines (arsenic, iron, quinine). To combat writers' cramp, we recommend, first of all, medical gymnastics and massage (active and passive movements of the muscles used in writing performed methodically, methodical writing exercises). *Galvanic* treatment also affords assistance (galvanization of the spinal cervical column and peripheral *galvanization* of the muscles affected).

Large Latin script is to be preferred to the German, lead pencils and goose quills to penholders; penholders should be large and made of cork. Special penholders are constructed (for instance those of *Zabludowski*, which are held between the index- and middle fingers). One should try also *Nussbaum's bracelet* (which makes the flexors and adductors superfluous in the writing process). Otherwise, the *typewriter* should be used.

As has already been mentioned, next in importance to writers' cramp is *pianists' cramp*, especially in females, who practice a great deal. Sometimes the hand is paralyzed, as if by spontaneous exhaustion, at other times the fingers can not move independently, or the cramp rigidly prevents the hand from striking the keys at all. *Violinists' cramp* affects the left as well as the right hand.

6. MASTICATORY SPASM (TRISMUS)

Spasms of the *muscles of the lower jaw*, innervated by the motor trigeminal, occur with relative frequency as a partial phenomenon of general diseases, rarely as isolated muscular spasms. In tonic masticatory spasm (*trismus*), both rows of teeth are so closely pressed against each other, that the voluntary opening of the mouth succeeds only in the slightest degree or not at all, and the jaws can not be passively separated. The masseters and temporales form clearly visible, prominent swellings; the patients can neither chew nor eat, so that food often has to be given artificially through gaps between the teeth, through the nose, or per rectum. Trismus is found in *tetanus*, *meningitis*, *epilepsy*, *hysteria*, *tetany*, *diseases of the pons* (for instance, progressive bulbar paralysis) and the *motor region of the brain*. As an isolated muscular spasm, trismus is excited by diseases in the region of the distribution of the *sensory trigeminal* (diseases of the mucous membrane of the mouth, caries of the teeth, periostitis, inflammations of the jaw bones, trigeminal neuralgia); it may also be the only symptom of a slight *tetanus*. Hysteric and epileptic individuals may under certain conditions present lock-jaw (trismus) as an isolated symptom persisting for a long time.

The clonic form of masticatory spasm, in which the teeth knock against each other (chattering of the teeth), is seen in chills and *nervous diseases* (for instance, paralysis agitans, hysteria). In connection with this, one should remember a peculiar condition of contraction in the tensor tympani, which is manifested subjectively, and sometimes objectively as well, by crackling sounds in the ear.

The **prognosis** of the disease depends upon the fundamental disease. In organic diseases, such as tetanus, meningitis, it is naturally, at least, doubtful; in hysteria, epilepsy, paralysis agitans, the spasm has only symptomatic significance.

Treatment.—At first the attempt must be made to discover the cause of the spasm, and for this purpose the *teeth, jaws and the cavity of the mouth* must be subjected to a close examination. When the spasms have a functionally nervous basis, bromides, arsenic, belladonna, the galvanic current are serviceable.

7. SPASM OF THE TONGUE (GLOSSOSPASM)

It is found only extremely seldom as a localized spasm and appears usually as a concomitant phenomenon of *hysteria, epilepsy, chorea, meningitis and psychoses*. Occasionally, it is evoked by *trigeminal neuralgias, caries of the teeth, stomatitis*. The spasm is manifested chiefly by clonic twitchings of the muscles of the tongue, the tongue either being thrust forward or against the palate. In the tonic spasm, it lies as a rigid body upon the floor of the oral cavity, usually pressed against the palate. Spasms in the muscles of the tongue, that appear upon every attempt to speak, and prevent speech (*aphthongia*), usually belong to the atypical forms of stuttering.

The treatment of the glossospasm depends upon the general disease that causes it. Iron, arsenic, bromides, belladonna frequently have a favorable effect.

8. SPASMS IN THE MUSCLES OF THE NECK AND NAPE

Spasms in the muscles of the neck may be of clonic, tonic and mixed character. The usual appearance is clonic twitchings with temporarily occurring tonic muscular spasms. The spasms affect either one or more muscles, and most frequently the following: the *sterno-cleido-mastoideus*, the three groups of fasciculi of the *cucullaris*, the *splenius*, the *scaleni*, the *deeper muscles of the neck*, more rarely the *platysma myoides*.

The spasms are characterized, according to the place and the degree in which they appear. If it be a matter of a unilateral sterno-cleido-mastoid spasm, the head is turned with the raised chin to the side opposite to the affected muscle. *Unilateral spasm* of the upper part of the *cucullaris* draws the head backwards, at the same time turning it to one side. By the *bilateral cucullaris* spasm, the head is literally hurled backwards; in spasm of the *splenius*, turned simultaneously to the side aud backward. Nodding spasms, which occur mainly in teething children (*spasmus nutans*), are really clonic spasms of the deep muscles of the neck; sometimes they are combined with rotary movements. They are frequently accompanied by nystagmus, strabismus, and blepharospasm.

The *degree of severity* of the spasm varies; occasionally it is so little pronounced, that there is difficulty in localizing it; then again, the most important functions are destroyed by its violence. The best result is obtained from complete rest, relaxation, diverting the patient's attention, and pacification, whereas the futile endeavor to overcome the spasm, just as all excitement, makes it worse, so that not even sleep can allay it. There is not much pain. The spasm may trouble the patient more or less constantly, or at intervals it may be of varying intensity; *consciousness* is not impaired; on the other hand, *psychoses* are frequent concomitant phenomena, as they seem to stand in close connection with the spasm.

In **differential diagnostic** respect, torticollis rheumaticus, diseases of the cerebral vertebræ, tic général and myoclonus must be considered. *Torticollis rheumaticus* is characterized by its great painfulness, which appears spontaneously, as well as upon pressure of the neck muscles affected, and upon the attempt to move these passively. In *inflammations of the cervical vertebræ*, there develops a flaccid (chronic), oblique position of the head, in which the corresponding sterno-cleido-mastoid stands out like a hard cord, and is quite frequently shortened (caput obstipum paralyticum). Difficulties arise in differentiating *tic général* from spasms in the neck muscles, if the latter be combined with a spasm of the facial muscles. In such a case, the apparently purposeful movements, echolalia and coprolalia, speak for the maladie des tics. In *myoclonus*, other muscular areas besides that of the neck are affected, and the spasm-like twitchings are of slighter intensity.

Anatomic investigation has shown no changes in the nervous system. Some investigators think it a disease of the cortex, others a disease of the cerebellum, and still others of the nerve nuclei of the cervical muscles. Various factors, such as the yielding of the symptoms to psychic influence, speak, at least in some cases, for a *psychogenic basis* of the disease.

Prognosis.—From a prognostic standpoint the condition is not favorable since the disease persists for many years or for a lifetime, even though remissions may occur. But now and again these spasms, even where they appear in high degree, are seen to disappear under various therapeutic measures. The spasms of the neck muscles observed in earliest childhood afford a far better prognosis, since they usually cease after the cutting of the teeth.

Treatment.—Physical and mental rest, occasionally the use of *antispasmodic and tonic* drugs (bromides, preparations of zinc, belladonna, iron, arsenic) are useful, and some physicians recommend *galvanic* and *faradic* treatment (anode on the n. accessorius, cathode on the muscle). Extraordinarily good results have been attained by *medical gymnastics*, either as exercise in holding the head still (*Brissaud, Feindel*) or as the "inhibition treatment" advised by *Oppenheim.* Some (*Wetterstrand, v. Reuterghen*) report favorable results from hypnotic influence. In case these various measures remain ineffectual, *operative intervention* may be considered. Sometimes, the stretching or resection of the n. accessorius has favorable results, whether they be permanent or temporary. Total *division of the tendons* of the nape muscles suggested by *Kocher and Quervain* also caused more or less improvement in a number of cases.

9. MALADIE DES TICS (TIC GÉNÉRAL)

This rather rare disease appears usually in *childhood*, and affects chiefly hereditarily *neuropathic* individuals, while direct hereditary transmission occurs only in isolated cases. The disease is usually introduced by twitchings in the *muscles of the face and neck;* to this twitching are gradually added movements that make an *emotional or purposeful* impression, for instance a throwing up of the head, reaching for the nose or beard, spitting, a wide opening of the mouth, jumping, dancing, etc., all of which are repeated again and again without variation. Such convulsive movements are distinguished from voluntary movements by their sudden brusque character, as well as by their lack of purpose. Speaking and breathing are frequently affected, as the disturbances described are accompanied by inarticulate sounds, that escape the patient. One hears cracking, smacking, barking sounds, or unconnected gibberish, also obscene expressions (*coprolalia*), here and there words (*echolalia*), or movements (*echokinesis*) are imitated.

It is usually a matter of a *plurality* of movements. By concentration of the attention on other things, the tic-like movements may be favorably influenced, so that the patients are actually sometimes in a position to control themselves through will power and carry on the duties of their vocation. In too violent a suppression, the convulsive movements appear with all the more force; they are also increased by psychic excitement. Some patients suffer from obsessions, and compulsory actions; must, for instance, count their steps in walking.

Course.—In the majority of cases it is a chronic, progressive disease, whereas in some, complete recovery takes place, in spite of long continuance.

Diagnosis.—The diagnosis is often difficult because of the similarity to other disease pictures. In *infectious chorea*, the muscular twitchings in contradistinction to those of the maladie des tics, are incoherent, have a continuous course; coprolalia and echolalia are missing. *Hysteric* persons sometimes present a disease picture very much like tic général. But here the beginning of the spasmodic phenomena can almost invariably be traced back to psychic causes; here also coprolalia and echolalia are absent. There are, moreover, *neuropathic persons* who suffer from a quite localized tic-like movement (such as shrugging of the shoulders, smacking the lips), which after a shorter or a longer time disappear. Such individuals can not be counted as belonging to the maladie des tics.

Treatment.—As much rest and seclusion as possible, light hydrotherapy, and, above all, medical gymnastics influence the condition of the patient most favorably. Gymnastic treatment (*Meige and Feindel*, *Oppenheim*) consists of exercise without apparatus and exercises in fixation of the individual parts of the body, that lead to suppression of the tic-like twitchings. Arsenic, hyoscyamin, and hypnosis are ineffectual.

10. PARAMYOCLONUS MULTIPLEX (MYOCLONIA)

Under this name, *Friedreich* in 1881 described a disease picture, that manifests itself in clonic twitchings of the muscles of the *trunk and the extremities*, while the *muscles of the face* remain unaffected. The twitchings are *lightning-like*, attack the muscles of both sides of the body with approximate equality, but not synchronously, since it rarely happens, that the same muscles on both sides of the body twitch at the same time. The twitchings, moreover, are unequal and irregular; violent oscillations alternate with others that are weak and scarcely perceptible; they are also arhythmic, following one another at intervals of varying length, so that at one time only a few are counted in one minute, at other times from 60–100. The disturbance always manifests itself as twitchings of individual muscles, which develop through muscular contraction, as if the muscles were shocked by a weak electric current.

The following muscles are most frequently affected: *the cucullaris*, *supinator longus*, *biceps*, *quadriceps femoris*, *semitendinosus*. Occasionally, also, the muscles of the face are affected. The twitchings disappear entirely during sleep, are often lessened by intended movements, but increased by psychic excitement. The *motor power* is intact; there are no muscular spasms. The *tendon reflexes* are usually increased; *sensibility*, mechanical and electrical *excitability* of the muscles and the nerves appear normal. The psyche is normal. A peculiar form of myoclonia has been described by *Unverricht*, *Lundborg, and others*. It is combined with epilepsy, appears as a familial disease, and attacks the muscles of the tongue, the throat and the diaphragm. In some cases, the decision whether a pure case of paramyoclonus multiplex is present, should be made with great care since definite cases have been reported which belong to hysteria and present symptoms of motor irritation much like those of myoclonia.

The **prognosis** of this chronic disease is unfavorable. Though there are occasionally remissions, the disease afterwards reappears. The cases of permanent cure are to be counted among hysteria. *Henoch's* chorea electrica is identical with myoclonia.

The **anatomic** examinations have so far given a purely negative result. In animals deprived of their parathyroid glands, *v. Wagner-Jauregg* has observed spasmodic phenomena resembling those in myoclonia.

Bromides, arsenic, galvanic and static electricity have a quieting effect. Thyroid gland tablets (1–3 daily) are supposed to have been occasionally effective. For cases, which form transitions to hysteria, psychotherapy comes into play.

11. PRIMARY ATHETOSIS

Primary athetosis (also called idiopathic or primitive athetosis) is a very rare disease, in which athetoid movements appear, though no sign of hemiplegia preceded them. It appears at any time of life, and usually affects both halves of the body symmetrically (athétose double). In some cases, the influence of cold or severe psychic excitement are adduced as ætiological causes. Whereas the athetosis occasionally rules alone in the disease picture, in some cases other nervous diseases are also found (epilepsy, mental deficiency). Usually the disease shows a progressive course; sometimes, too, it remains stationary. Anatomic examination has usually given negative findings. At any rate, this disease must be most rigorously differentiated from the diplegia spastica infantilis, that is combined with athetosis.

SUPPLEMENT

Ménière's Disease (Ménière's Symptom-complex)

Ménière's disease is an affection that manifests itself in *difficulty of hearing, tinnitus aurium, vertigo and vomiting*, and either suddenly attacks individuals whose ears are sound (Ménière's apoplexy), or is associated with acute and chronic diseases of the ear. The apoplectic form, which is by far the less common, is found in diseases such as *syphilis, parasyphilis, arteriosclerosis, nephritis, leucœmia;* occasionally, also, in connection with a *traumatism* (contusion of the skull, injury of the labyrinth, caisson disease). Ménière's symptom-complex, the more common form, occurs in the most varied affections of the *middle and the internal ear* as well as in *diseases of the auditory nerve.* Here belong especially otosclerosis, chronic adhesive processes of the middle ear, chronic and less frequently acute purulent inflammations of the middle ear, auditory nerve diseases, diseases of the external ear (cerumen) and of the labyrinth; here we must also mention epidemic cerebrospinal meningitis, as well as poisoning with quinine and salicylic acid.

In most cases, the *deafness* is of a rather high degree, but in some cases only slight. The *vertigo* is usually very violent, producing a feeling of turning or being hurled around, during which the direction of rotation sometimes changes, sometimes remains the same; if the vertigo appears in very intense form, the patients fall prostrate or seek to find a horizontal position. The patients nearly always suffer from continuous *tinnitus aurium*, which is still further increased by the attacks. Ménière's disease is also often accompanied by *vomiting*, occasionally too by painless diarrhœas. Loss of consciousness is rare. As concomitant symptoms are found nystagmus, swimming of objects before the eyes, darkening of the visual field, rushing of blood to the head, outbreaks of perspiration.

We must regard a disease of the *labyrinth* or of the *auditory nerve* (hemorrhages, infiltration), as *basis* of Ménière's apoplexy. In the accessory (secondary) Ménière, one finds pathological processes in the *middle and internal ear, the auditory nerve* and its intracerebral ramifications. The deafness must be traced back to changes in the middle ear and the labyrinth, whereas the whirling vertigo is connected with the semicircular canals.

Differential Diagnosis.—Sometimes it is difficult to distinguish the apoplectic Ménière's attack from *cerebral apoplexy*. Though the insult in Ménière's attack does not lead to very deep disturbance in consciousness, it is combined with sudden deafness, tinnitus aurium, and vomiting. Rather often one has an opportunity to differentiate Ménière's symptom-complex from the various *forms of vertigo* (vertigo due to the eyes, intoxication with nicotine, quinine, salicyl, vertigo in nephritis, diabetes, arterio-sclerosis, neuroses, parasyphilis). During the attack of Ménière's vertigo, the patient usually lies in bed, complains of tinnitus aurium, vomits repeatedly, clutches anxiously at objects. In the periods between the attacks one is limited to the statements of the patient. By the name "*pseudo-Ménière*" (*v. Frankl-Hochwart*) attacks of vertigo, tinnitus aurium, and vomiting in persons with sound ears, are designated. They are usually partial phenomena of an hysteric or epileptic attack.

In **treatment**, general therapeutic measures must be taken into consideration, besides otological treatment. Hydrotherapy, climatotherapy, massage, and head galvanization must be mentioned here as most important. Of drugs, bromides, iodides, and quinine are used; bromide of sodium in daily doses of 2–3 g., iodide of sodium daily 1/2–1 g. Treatment by quinine (daily doses of 1.0 g. for 2–3 weeks) which was used by *Charcot*, is now, for the most part, abandoned.

VIII
PSYCHASTHENIC STATES

BY

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INTRODUCTION

The diseases we are about to treat under this heading must be regarded from a uniform point of view. It is true that apparently the diseases known as *hysteria* and *neurasthenia*, when seen in their most characteristic forms, have scarcely anything in common. An anxious neurasthenic, living in seclusion, who shyly conceals his obsessions and his anxieties from the world, is apparently so unlike an explosive, irritable, hysteric, who by loud and persistent lamentations and by enumeration of his numerous woes wishes to become the center of general sympathy, that it may seem strange that they should be considered as types of one and the same morbid tendency. Upon investigation of a great number of cases, however, it is obvious that transitional conditions occur so often, that neurasthenic affections are so frequently combined with hysterical stigmata, and hysteric convulsions so frequently associated with general neurasthenic complaints, that there can be no doubt of the close relationship between the two affections. The more frequently we look for the symptoms of the one disease in the other, the more frequently we find that the sharp lines of distinction disappear. Not even the multiformity of hysteria, or what has frequently been called its protean changes, to which especial significance was formerly attached, is a regular manifestation of hysteria. Besides cases that show a new symptom every day, there are also those that for years stubbornly show the same uniform symptoms, as well as cases of monosymptomatic hysteria, and, on the other hand, cases of neurasthenia with affections that vary from day to day.

Even though a sharp line of separation is not possible, it is feasible for didactic as well as therapeutic purposes to adhere to some distinction between the *clinical pictures*. Only by the use of a certain schematic method is it possible to present the clinical picture of diseases in a manner that is true to life, without destroying by a mass of details the clearness of the drawing. If, then, I attempt to portray hysteria and neurasthenia separately, it is only that the typical clinical pictures may be distinguished; with regards to this schematic presentation, however, one should never forget that it is concerned with two phases of the same disease.

Hysteria transforms representations (ideas) into *physical manifestations;* neurasthenia transforms physical manifestations into *emotions*. The hysteric is afraid of paralysis, and his arm is paralyzed. He feels a pain in his throat, and the vocal cords refuse to work. The neurasthenic feels a pressure in the region of his stomach; immediately the ghost of "cancer in the stomach" rises before him. He finds that his memory is not so good as it was, and fears softening of the brain. The thought of the hysteric moves in *the past* and *the present*. He portrays all that he has already experienced, and whatever is troubling him at the moment. The maladies of the neurasthenic, on the other hand, are troublesome, only in so far as the mournful present is to be followed by a more hopeless *future*, or in so far as he has constantly before his eyes the development of his as yet slight illness into serious and fatal diseases. It is easier to influence the hysteric because he likes to display his troubles; the neurasthenic, on the other hand, broodingly hides his most serious affections in the depths of his being, because he avoids what he considers a lack of intelligence on the part of the healthy.

These are—omitting many manifestations—a few of the differential features. They lie, without exception, in the *psychic realm;* and I have purposely brought them to the fore, to prove, that in neurasthenia, as well as in hysteria, we are dealing with *psychic diseases*. They are generally called *neuroses*. This appellation serves a purpose in soothing the patient. He and his relatives dread the very idea of *mental* disease. The physician, however, must not be misled by this fear to a non-recognition of the fact, that the most important and essential symptoms of these diseases are of a purely psychic character.

Cramer defines nervosity—our psychasthenic states—as a disturbance of the general harmonious functional capacity of the brain, which comes to be expressed as a certain insufficiency; and attempts to divide these diseases sharply from real mental disorders. I really cannot endorse this conception. The criteria given by him, do not, in my opinion, hold good. Changes that are detectable anatomically are also absent in many obvious cases of psychosis: and in nervosity we find also—and not seldom—the formation of defective or erroneous judgments following emotional disturbances.

Psychasthenic states belong to the broad *borderland* between mental health and disease, and are distinguished from many forms of psychosis, as, for instance, from the lightest cases of manic depressive insanity, of constitutional depression, and of degenerative states only by the slighter degree of their manifestations. We may calmly recognize fully that the psychasthenic states, the symptoms of which lie mostly in the life of the soul, or depend upon psychic life, must be considered as *slight forms of psychic disorder*; then we shall be more likely to do them, and our patients, justice.

1. HYSTERIA

Definition.—The manifold characteristics of hysteria, in the course of centuries, one might almost say of thousands of years, have been observed so minutely and carefully, that a description of the symptoms can scarcely include anything new. Mere external description, I mean—since from a description to an understanding of how the symptoms arise, is a long step—and in spite of all that is new and important, in spite of all that has been brought to light by the work of *Charcot* and his school, especially of *Pierre Janet*, and the Germans *Voigt*, *Moebius*, *Freud*, *Binswanger*, and *Oppenheim*, to name only a few, we have not yet passed beyond this stage.

All these workers attempt to reduce the manifestations of hysteria to a formula, to explain them from a uniform point of view. But even if that had been successful, we would not have reached our ultimate goal. This goal consists in the explanation of why and how arises the uniform *mental condition*, that constitutes the symptoms.

Whether we shall ever succeed in reaching this high goal, must, for the present, appear rather questionable. The hope that *anatomy* and *chemistry* may be of assistance to us is very slight. And so it is alloted to us, first, to attempt to reach a more uniform conception on the basis of an analysis of symptoms, an attempt that is as old as the name itself (hystera-uterus). This attempt makes it sufficiently clear, as to how the disease was explained: the desire of the uterus for satisfaction was at first supposed to be the cause for the appearance of the symptoms, and the origin of the disease, which idea was later replaced by the more medical conception of a disease of the uterus and its adnexa.

Thousands of years have passed, and we must not conceal from ourselves the fact that we have again reached the starting-point. It is no longer the uterus itself that rises and falls in the body, but *sexual desires*, satisfied and unsatisfied, that, according to *Freud's* theory, gives rise to the serious symptoms. For him the manifestations of disease in hysteria represent "sexual activity." According to his conception, sexual experiences, desires and ideas are consciously or unconsciously *suppressed*, and thereby *converted* into physical manifestations.

I consider this view, which cannot be further discussed here, as erroneous, and the close inquiry into sexual matters as a very questionable procedure. But even if Freud's theory were correct, no insight into the question would be gained: why do such injuries give rise to hysteric symptoms, and why only in certain individuals? We should then be obliged to assume a special peculiarity, a definite predisposition.

It is certain that *diseases of the uterus* and its adnexa can *not* be the cause of hysteria. This idea was given its death blow by this fact alone, that hysteria has been observed in *men*, also, and very frequently in women who have *sound* genital organs. All attempts to bring hysteria into causal relations with coarse physical manifestations or diseases of the sexual organs, as of the rest of the body, have proved futile.

If we attempt to describe the symptoms of hysteria briefly, one phenomenon meets us again and again: *the false relation between stimulation and reaction.* Either the stimulation is perceived too strongly, too weakly, or not at all, or the reaction is exaggerated, or too slight, or totally absent. This formula explains narrowing of the field of vision, as well as anæsthesia, and hyperæsthesia; it explains the paralysis of an arm by a blow and the paralysis of speech as the result of terror: it fits in with hysterical seizure as well as with hysterical asthma, with the pleasure found in sensation, with egocentric heedlessness of others, as with the exaggerated altruistic joy in sacrifice, all characteristics of the hysteric character.

The strong *emphasis of the emotional moment*, gives something unaccountable to every stimulation. The *emotional excitability* is not the same in every case. An occurrence, which at one time makes a deep impression that is overcome with difficulty, at another makes none at all, according as to whether either the *attention* or the *emotional readiness*—this holds good especially of the relations between man and man—is brought into play.

I consider the *predisposition to be* easily *influenced*, which is a peculiarly striking symptom of hysteria, as only a partial phenomenon of the emotional excitability. The patient can be so easily influenced, whether for good or for evil, because everything in him calls forth unusually vivid feelings of attraction or repulsion. *Autosuggestability*, also, is most intimately connected with this excitability of the feelings.

Not the *strength of the reaction* to a stimulation alone, but its *manner* as well, is characteristic of hysteria. This is displayed pre-eminently in the fact that *ideas and sensations are transformed into physical manifestations. Möbius* has classed those diseased changes in the body, as hysterical, which are caused by ideas. This conception is too broad, inasmuch as we are not justified in classifying as hysterical every physical manifestation arising from an idea. I call attention, for instance, to "feeling sick" and vomiting, as a consequence of ideas that excite disgust. And it is too narrow, since we find physical phenomena in hysterics which we can not trace back to ideas. It can not be doubted, however, that *Möbius* with his explanation came very near to the correct solution. *Sommer's* proposition, to call hysteric phenomena *psychogenous*, has much in its favor. For this reason, if no other; because in the eyes of the laity, and, sad to relate! occasionally, even of physicians, the designation of a symptom as "hysterical" is accompanied by a *prejudice* overcome only with difficulty, the prejudice, namely, that it connotes shamming and a sexual cause.

In spite of all these utterances, we have not as yet approached much nearer to the nature of hysteria. Experience teaches that hysteria develops on the ground of a *psychopathic predisposition.* We almost always find serious disturbances of all sorts, in the ancestors; and—a proof of the seriousness of the disease—the *descendants* of hysterical patients usually have still more severe diseases. Education also has a strong influence, bringing about the disappearance of the old symptoms, and the appearance of new ones through psychic impressions. Here again we face the problem, as to why *endogenous predisposition*, given a similar education, manner of living, and lot, should in one case give rise to grave hysteria, and in another to none at all. No theories really give us an explanation of this. Neither *Janet's* "limitation of the field of consciousness and cerebral insufficiency," nor *Sollier's* vigilambulism, nor *Hellpach's* increased docility, nor *Jelgersma's* under development of the central nervous system, helps us to solve the riddle of hysteria, though I do not deny that the setting up of each new theory serves for new investigations, and hence helps to further the solution of the problem.

It is probable that we shall be for a long time without a conclusive explanation of the nature of hysteria. I shall confine myself therefore, to the attempt to state, in a brief formula, the *fundamental symptom* of hysteria, from which, in my personal opinion, all the manifestations may be deduced.

Hysteria is a disease established on an endogenous psychopathic foundation, which is characterized by the false relation of the stimuli, especially those of an emotional nature, to the reactions, which not infrequently display the peculiarity of converting ideas and sensations into physical manifestations.

Ætiology.—Hysteria is a disease, that presupposes a psychopathic predisposition. But it would be a mistake to expect by reason of this fact that a very great number of patients suffer as a result of heredity. Our own statistics as to the previous life of parents and ancestors are too insufficient and untrustworthy, to furnish absolute proof in each case. But we shall not go far astray if we estimate that 70–80% of the patients suffer through inheritance. In many, the inferior resistance of their disposition may be due to injuries of the germ, in many, perhaps, to diseases which, during intrauterine life, or in their earlier years, lead to constitutional injuries. Whether a definite kind of hereditary taint is influential on hysteria, is not known, but one thing I have often noticed, that light hysteria of the parents reappears in the children in increased measure.

Women are more frequently hysteric than *men*. All observers are agreed as to this, the proportion given varying from 1 : 5 to 1 : 10. The number of male patients is increased by the number of cases of hysteria following accidents (cf. discussion thereupon). But, as a fact, men seem to be subjected to the neurasthenic women to the hysterical form of the disease.

Race, too, seems to be of importance. At least, the serious cases, especially those accompanied dy convulsions and subsequent delirious conditions, are apparently more prevalent among Latin and Slavic, than among Teutonic peoples. Among the Jews, too, in my own experience, cases of hysteria, though mostly in vaguely defined form, occur with unusual frequency.

That we are not mistaken in the supposition that hysteria is dependent upon a congenital predisposition, is proved by the countless cases in which *very early in life* manifestations appear which must be designated as hysterical, I saw a child of two, several of three and four years old, with unmistakable hysteric symptoms. In these cases it is usually possible to prove that the relatives have been particularly *careful* of the children, because of physical diseases or of one kind or another of congenital debility. It is certainly not due to chance that among hysterical children as among hysterical adults a surprisingly large number of cases occur in which the hysteric is either an *only child* or a child who, born after a long lapse of time, has been spoiled not only by its parents, but also by its elder sisters and brothers as well. An only daughter, too, among a number of sons, and vice versa an only son among many daughters is also subjected to hysterical predisposition. Any slight physical ailment is the occasion of anxious observation, in which the child soon participates. *Attention* is called to its own body, instead of being *drawn away* from it, and the *predisposition* that may until then have been *latent*, is made *manifest.* If such a case is not made the object of a definitely designed education and treatment, there is established a clearly defined hysteria, one that rules the entire life. I lay special stress on the damaging influence of a faulty *education*, for the proper comprehension of hysteric phenomena.

The time in which the manifestations are most likely to appear, is the *period of development.* I do not believe that the usual *chlorosis* of young girls at this time, represents an actual cause, perhaps not even an exciting cause. In many cases chlorosis may well be regarded as the expression of an inadequate physical predisposition that can not overcome the dangers of the period of development.

At the same time occurs the first budding of *sexual sensations*, and therewith the danger of *masturbation.* I shall not here enter more closely into the relation between sexual life and nervous diseases, because I shall have to discuss it in detail in the chapter on *neurasthenia,* and therefore shall remark only, that in my personal experience—and I can certify, that I have paid particular attention to this very circumstance—sexual life exerts no dangerous influence, worth mentioning, on the nervous system. In those cases, in which we believe that we notice such influence, it is to be traced back to the *ideas*, associated by tradition and prejudice with sexual life, but which, per se, have nothing to do with it.

The *diseases of the sexual organs*, too, are to be stricken from the list of the factors concerned in the causation of hysteria. A careful gynæcological investigation reveals often enough displacements, adhesions, atrophic and other processes. But we find the same phenomena in women who are not hysterical. If the gynæcologist removes the *local* troubles, the hysteric symptoms occasionally disappear, but they would have disappeared likewise, if another workable suggestive method of treatment had been adopted. With a few exceptions, even gynæcologists have come now to the conclusion, that the fact of their former busying themselves excessively with the uterus and its adnexa in cases of nervous diseases was injurious.

Only one cause deserves special attention, because, more than all others, it is adapted, as if with one blow, to make a serious disease out of what was until then a harmless hysteric predisposition, and that is *emotion* (Affekt). In enunciating my conception of hysteria I have already pointed out the importance of *emotional irritability*. This is a manifestation of the disease, a preliminary condition, the importance of which is especially shown, when a really serious emotional disturbance strikes the susceptible hysteric. The peculiarity of his mental make-up is such that not every emotion works with equal force. Especially injurious are events that happen *suddenly* and unexpectedly, which at the moment of their occurrence cause embarassment, terror, etc., as thereby quiet deliberation and calm consideration are prevented and the patient harmed. In the same way also chronic, that is to say, *persistent emotions*, such as cares, the feeling of being neglected, fear of examinations, failures, have a similar injurious influence. Among all the causes that can educe from the latent predisposition a manifest disease —*agents provocateurs*, *Charcot* calls them—the emotions certainly deserve the first place, and I do not think I am mistaken, if even in physical diseases I attribute greater importance to the cares concerning one's own body, and anxiety as to a fatal outcome, than to exhaustion of strength.

Therefore, I shall not take into account all *psychical diseases*, with which hysterical phenomena are associated. They permit us to recognize the latent hysterical phenomena in the patients only by cruder symptoms. If we were to enter more carefully into the *previous life* of the patient, we would seldom fail to elicit in his former history, signs of nervous predisposition, which, because of their slight obtrusiveness, have remained unnoticed, but which prove that hysteria was there before.

An exceptionally extensive experience shows the importance of *imitation* as the causative factor of hysteric manifestations. This danger is increased by the hysteric's *craving for sensationalism*, which is attracted by everything unusual, especially by morbid phenomena. One may count with considerable certainty on the fact, that a case of real chorea minor brought in association with a case predisposed to hysteria causes restlessness deceptively like chorea, if the association is allowed to last any length of time; just as a serious hysterical attack in a hospital ward seldom remains an isolated case. It is clearly not a matter of *voluntary* imitation; evidently the impression, that calls forth the interest of the patient, becomes very quickly a sensation, which causes involuntary movements, to which sensation the example set by the first patient gives a more exact form. The cause of a single symptom or of a group of symptoms may often be traced back to imitation, but I exclude the possibility that the *disease* hysteria can be the result of looking at a hysteric or epileptic fit.

Morbid Anatomy.—A *pathological anatomy* of hysteria is absolutely lacking, and we have little reason to hope that the future will give us clear and unambiguous information as to the character of the disease. The manifestations of the disease are frequently so transient, undergo such serious changes under psychic influences, that rougher anatomical changes could not be the underlying basis of the disease picture. We may more reasonably hope to find an anatomical basis for the *constitutional predisposition.* But this hope, too, is very slight; at any rate, no way has been as yet discovered o bring us any nearer to our goal.

Symptomatology.—*The hysteric character.* We designate as the *hysteric character* an individual peculiarity which seems to be well known even to the laity, a strange mixture of *coolness* and *excess of emotion*, of *sweet friendliness*, and *enmity*, *capriciousness* and *obstinacy*, *untruthfulness*, and *egoism*, and *maliciousness*, not at all a pleasing picture. But we must not overlook the fact that this peculiarity is nothing specifically hysterical, no manifestation of hysteria, but of *congenital degeneration;* we find the same characteristic peculiarities in the non-hysterics, even if in hysterics we do generally meet them most clearly defined, in full bloom, so to speak. There are, besides, very many hysterics, whose character—it being quite impossible to prove the existence of all, or even of a large part of these unpleasing traits—shows a wholly sympathetic personality.

Hence is explained why many writers, as *Wollenberg* and *Loewenfeld*, deny outright the existence of an hysteric character. I should not go so far. I too consider the peculiarities described—*Cramer* speaks even of decided *ethical defects* and a certain *moral depravation*—as manifestations of degeneration, which springs from the same underlying conditions that produce hysteria. But we find some of the above individual traits in nearly every case of hysteria, and if they do not all develop to their fullest extent, the cause lies more often in external, especially in *educational* influences, than in the non-existence of the peculiarities themselves. They are too much grounded in the very nature of hysteria not to be almost always present, either latent or slightly manifested.

Nothing particular is to be said regarding the *development of the intellect* in the hysteric. This is not wholly indifferent, in so far as the form of the disease, and the kind and degree of its manifestations, are influenced by intelligence. The less intelligent the patient—this is a rule subject, however, to many exceptions—the more uniform the clinical picture, the more limited to spasms, sensory and motor symptoms; the more intelligent the patient, the more manifold and changeable the disease pictures.

Almost always the development of the intelligence in hysterics gives the impression of *one-sidedness*, which is nothing more than a consequence of the vivid *emotional tone* which accompanies every occurrence. The sensitive receptiveness makes the patient more ready to comprehend, he becomes *inspired* more easily than others; the ability afterwards to *revitalize* these feelings, to live in others' emotions, in others' thoughts, permits him frequently to grasp the point of a situation with extraordinary acumen. So the hysteric may soon become the leader in a great movement, which sprouts first, possibly, in the great mass of the people, or may perhaps become the passionately enthusiastic follower of an idea, or the terror of those about him, whose errors he everywhere seeks out and recklessly exposes with this talent of one-sided attention.

If the achievements of hysterics fall far behind those of equally gifted normal men, the reason lies in their tendency to be *switched aside*, and their one-sidedness. Work that is carried on quietly conscious of its aim, which gives proper attention and necessary industry to what is less fascinating, but essential, represents but little the temperament of hysteric predisposition. The persistence with which the hysteric pursues an aim which appeals to his feelings fails in relation to everything that leaves him unconcerned. So we find, side by side, according to circumstance, joyful effort and an absolute refusal to work, yet both are conditioned by the same phenomenon, *strong emotional excitability*.

The activity of the emotions, the *affective susceptibility*, explains to us most of the traits of the hysteric character. The patient is *exceedingly sensitive* and *easily hurt*. Every trifle excites him, and if his own person is concerned—and where does the hysteric fail to find some relation with his own person—he understands every harmless remark as an insult, every slight offense as a most serious outrage. This sensitiveness may become an unbearable torture for those about him. To mention an example: his relatives ask him how he has slept. Then he complains because they keep reminding him of his illness and annoy him with superfluous curiosity, or believes that they wish to show him that they put no faith in his complaints of sleeplessness. The family, therefore, ceases questioning: then the patient is offended, because they do not show sufficient interest in his condition even to ask about it, or because they prove, by ignoring his sufferings, that they consider it all sham. The usual result of the strong emphasis upon the ego, is that the patient becomes estranged from those surrounding him, because he heedlessly places himself in the foreground, and shows to others none of the *delicacy* that he demands for himself and his troubles.

Yet it must not be considered contrary to this idiosyncrasy if simultaneously, the same patients are able to perform the greatest possible *self-sacrifice*. This is due partly to the *possibility of inspiration*, with which they grasp an idea. That these ideas are not always the most common ones, is natural enough. Even in healthy people an impression recurring daily becomes so blunted as to be finally indifferent. How much more must this be true of hysterics! They are enthusiastic only about something *unexpected, unusual, even forbidden.* But when they do become interested, they seek to express their feelings in actions.

And all the more gladly, if as a reward for the exaggerated self-sacrifice are awarded him, the *recognition and admiration* of others. Not that the applause of those about him is always necessary. Often enough, he is quite content with the inner satisfaction that he finds in sacrificing time and trouble, and giving up his own comfort. At times, too, he has the satisfaction of feeling that he has accomplished what others could not or would not do. The patient does not even realize that it is simply *egoistical* motives which frequently determine his actions, when the outward appearance is that of mastery over every egoistic feeling.

This peculiarity touches most intimately the inclination of the hysteric, *to play a rôle.* It makes little difference, whether the rôle be that of a patient sufferer, tortured by fate, or of a political martyr sacrificing herself for an idea. But one is unjust to them in supposing that they play this comedy after calm deliberation, and with accurate calculation of the effect to be attained. It is due rather to a *strong desire to pose*, to live in strange situations and sensations, which is stronger than sober consideration and sound will-power.

This peculiarity is subserved by a certain *unreliability* of the *memory.* Here too, emotional excitability must be adduced as the most reasonable explanation. The patient becomes so fixed with an idea, that every wish, every possibility seems to him either immediately realized or near realization; he loses completely the ability to distinguish truth from falsehood, wish from fulfilment. This gives to his conduct the appearance of unartificial *truth to life*, which has often helped these men to successes, that to the distant observer must seem almost incomprehensible.

Even critical people are deceived, when the patients scatter about the most serious suspicions of those surrounding them, or in moving words, complain of the lack of comprehension in their relatives. Often enough, an actually existing *lack of understanding* on the part of the family, is the starting point of conscious *exaggeration.* But while the patient exaggerates, the ground beneath his feet gives way. The vividness of his *phantasy*—again the result of the strong emotional tone—changes every wish into reality, every fear into an accomplished fact. So fear of paralysis gives rise to paralysis, fear of an illegitimate pregnancy, the conviction of being pregnant.

How it happens that these feelings are *transformed into physical phenomena*, that the breasts begin to swell, the linea alba colors, the body becomes rounded, and even menstruation fails to appear, evades our knowledge. But experience teaches only too frequently, that every idea may be physically mirrored forth in serious changes, paralyses, anomalies of vessel innervations, swellings, hemorrhage, cessation of menstruation, persistent vomiting, diarrhœa, etc. The increased excitability makes of the patients body an instrument, whose discord no one can feel more strongly than the patient himself.

The *autosuggestibility* of the patient may easily be traced back to increased emotionalism, even if we can explain but roughly the influence of emotional life on the physical organs by the dependence on the innervation of vessels. Autosuggestibility is but one side of *suggestibility*, in which again we find the fundamental trait of strong emotional excitability. Whatever makes the greatest impression upon our emotional life, makes also the greatest impression upon our thoughts. This general psychological experience makes us understand why the physician, and, we may safely say, the quack as well, has such unbounded influence on the phenomena of the disease, as long as he possesses the confidence of the hysteric. On the other hand, the slightest slip, associated with a beginning mistrust of the physician's skill, is sufficient to destroy all further progress towards recovery.

The hysteric knows no *objective criticism;* he is always *subjective;* he cannot keep to the golden mean, but always moves in *extremes*, even in his *attractions* and *aversions*. And woe to him for whom he has an aversion. If he is offended, or considers himself offended, he does not take it at all harmlessly. The strength of the inner excitement calls out in him the desire for satisfaction, the wish for revenge. Here too, suggestibility, the intensity of emotions and the capacity to easily adopt the feelings of others plays a deleterious rôle. The patient *distorts*, consciously rather than unconsciously, the words of his opponent. A suspicion that he has—and cause is easily found in his mistrust—becomes a fact, nor does he hesitate to *exaggerate*, even consciously, his suspicions.

Very often another peculiarity is associated with this procedure: the *desire for sensationalism*. The patient enjoys his strong emotions, can not dispense with them, and seeks them, where they do not present themselves. So the general consternation brought about by anonymous slanders, the branding of an honest man by false charges, may become a necessity to him, as much as the wish to be pitied because of a serious misfortune, or a painful illness, or a more intimate anxiety regarding an operation, which he may bring about, quite unnecessarily, by complaints with respect to simulated pains and false temperature records. It is certain, that in the majority of cases, the flirting and adultery of hysteric women, is caused less by *sexual excitability*, which in hysterical women is but little developed, than by the *desire to satisfy their need of sensationalism*. Not an instance of horrible misfortune is terrifying enough to such patients not to be utilized, no sight so repellant that they would avoid it, even if afterwards they can not do enough to express their sympathy or disgust.

I should not like to close this description of the hysteric character without a warning against a misunderstanding that we meet but too often. Though the patient does occasionally exaggerate, and even directly deceive, though he is fond of sensation and slander, capricious and exacting, though he may torture his physician and relatives to the utmost, and repay all their care with blank ingratitude, we must never forget that he is sick, that all the traits described rise in a *diseased, increased excitability of the emotions*, and that he is of all the sufferers the one most tortured by his unfortunate predisposition.

Disturbances of Sensation. Scarcely a case of hysteria is without *disturbances of sensation.* Those very complaints about pains that are narrated so vividly; pains that now change, now remain stubbornly in the same spot, give the characteristic color to the disease picture. Since the work of *Briquet* and *Charcot*, many investigators have occupied themselves with disturbances of sensations in hysteria, and always have found more or less clearly defined changes in cutaneous sensations. Only in most recent times, has it been asserted, first by *Böttiger*, then by the Paris school, especially by *Babinski*, that no sensory disturbances occur, unless physicians by their *search* for such disturbances have produced them artificially.

I hold that this view is incorrect to this extent, that sensation disturbances have been proved to exist in patients whose cases have never before been investigated from this standpoint, generally to the great astonishment of the patients themselves. That it is not a matter of the result of such investigation is proved by the peculiar arrangement of the disturbances, and by the difficulty of changing, with the help of suggestive questions, the distribution with respect to the anæsthetic parts. The real gist of the doubts of *Böttiger* and *Babinski* lies in the conviction shared by me, that an organic anomaly is not concerned in this phenomenon, but that it must be regarded as purely *psychogenetic.* It is not, however, as Böttiger assumes, suggestion by medical investigation and inquiry that gives rise to the changes or disappearance in sensation, but processes unknown to us.

Naturally, the method of investigation may give rise to a disturbance in sensation; but then, just this *artificial* suggestibility is very significant diagnostically. I have occasionally used the suggestibility of the patients in order to establish a diagnosis in serious cases by the changes in cutaneous sensibility taking place before my eyes, i. e., in convulsions, when the difficulty lay in distinguishing between epileptic or hysterical, or where I had lacked opportunity to observe them. For this purpose I proceed thus: I touch with the point of a needle, with unequal force, two spots which I have formerly proved to be of equal sensibility. In hysterics there soon appears the idea that they have less sensation in the spot less strongly touched, and after a short time, one can prove definite lack of sensibility for this spot. I wish to emphasize that only the positive result of this test, which is quite harmless for the patient, points towards hysteria, but that the negative does not militate against this diagnosis.

The *test of sensation* is best undertaken in this way: Touch with a soft hair brush, as evenly as may be, alternately, symmetrical parts of the body, or, in case there is no difference between the two sides, the extremities, distally and proximally. The investigation is then repeated with a pin, since quite often the sensation for delicate *touch* is perfectly normal, where *pain sensations* are different. If one holds a rather heavy needle loosely between two fingers, it is quite easy to touch the tested parts uniformly. We may forego, generally speaking, the tests for sensations of heat, or cold, or the electric current. I have found that the trouble spent on these reactions did not pay.

Complete loss of sensibility in the body occurs rather seldom. A complete sharply defined *hemianæsthesia*, is also rare (Fig. 208*a* and *b*). By careful investigation one can usually find in the anæsthetic part of the body a few island-like spots where the patient feels touch and the prick of the needle. A characteristic attribute of the anæsthesia of hysterics is a *distribution according to organs*, as the laity conceives them. The arm, the forearm, the hand, the breast, the back, bandage-, glove-, or stocking-shaped places are anæsthetic (Fig. 208*c* and *d*). This disturbance is distinguished from the organic, in that it has no connection with nerve distribution.

An exact distinction between anæsthetic places, and those, in which sensibility is merely decreased, is usually impossible, and always superfluous. The like holds true even for a careful distinction from the zones of *hyperæsthesia*, which occur occasionally, next to, or among the anæsthetic regions (Fig. 208*e*). The hyperæsthetic places have a significance merely because they are easily mistaken for diseases in the organs lying beneath them. The pressure on the skin causes pains which are localized deeper by the patient, thus giving rise to false diagnostic conclusions, to the assumption of neuralgias, and, under certain conditions, of serious inflammatory processes.

Disturbances of sensibility affect not only the skin, but the *mucous membrane* as well. Especially characteristic in this respect is the loss in sensibility of the *sclera* and *cornea*. While, some people recoil from a touch on the sclera, or at least close their eyes, and while a touch on the cornea produces these movements in almost everybody, this touch in the hysteric frequently calls forth no defensive motion of any kind. The *mucous membrane of the nose*, too, which in a healthy person, is very susceptible to touch and slight needle pricks, shows at times, corresponding to the anæsthetic half of the body, a decrease of the defensive reflexes. The anæsthesia of the *palate* I consider less important; in hysterics, who smoke or drink, the *retching reflex* is sometimes very strong, and, on the other hand, it is absent in many non-hysterics.

Among the *sense-organs*, the eye must be regarded as most important. *Sight* is sometimes wholly lost in both eyes, sometimes in one only: as a rule, in the latter case, on the side of the body affected with cutaneous anæsthesia. Still more frequently, there is a high degree of *narrowing of the field of vision*,

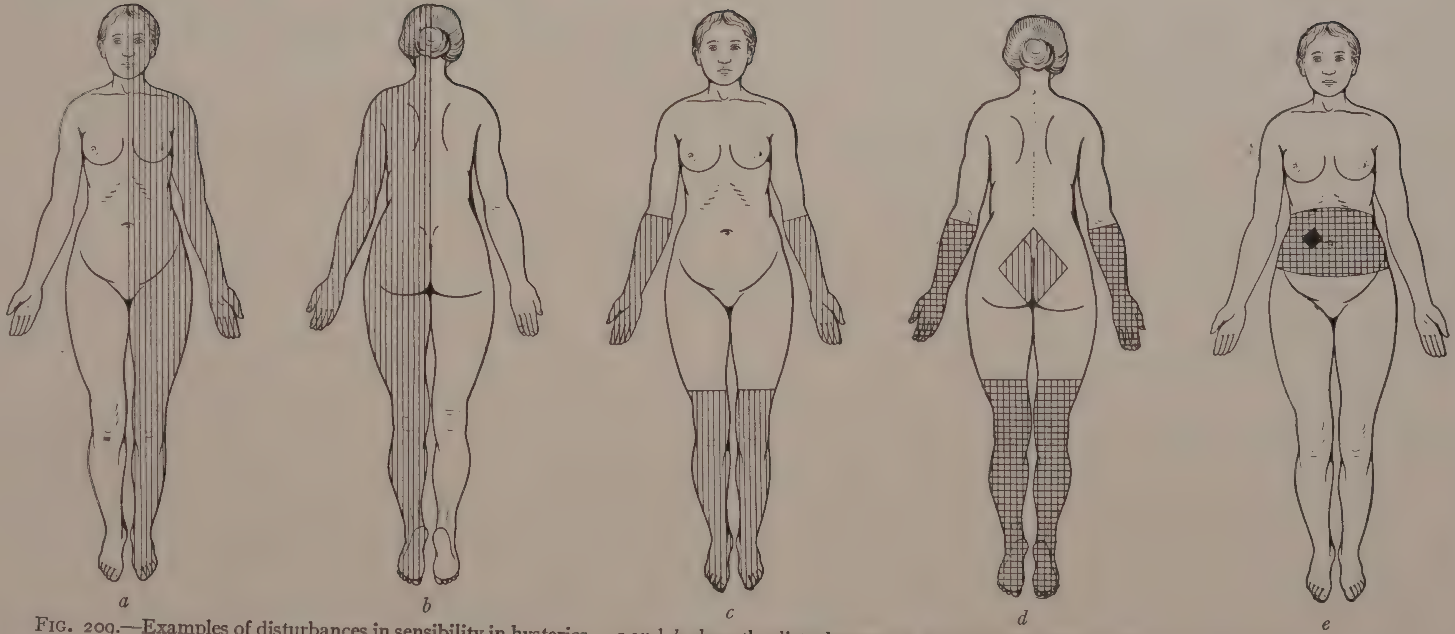


FIG. 209.—Examples of disturbances in sensibility in hysterics. *a* and *b* show the disturbances on the front and back of the same person, also *c* and *d*.

▯ Normal sensibility. ▥ Anæsthesia. ▦ Hypæsthesia. ■ Hyperæsthesia.

which, as a rule, is concentric for all colors and usually bilateral (Fig. 210), even if somewhat less strong, generally, on the anæsthetic side. The field of vision may in some cases, be quite tube-like, and yet the patient may orientate himself in the room and recognize his surroundings. I have even seen a patient with a visual field scarcely worth mentioning skate on a pond without any difficulty. This is important as furnishing the key for the comprehension of the symptoms. While in retinitis pigmentosa or tobacco amblyopia a high degree of narrowing of the visual field robs the patient of the power of finding his bearings in a room, the hysteric is, for the most part, not disturbed by narrowing of the field of vision, and generally does not know anything about it, until it is disclosed at examination.

One can only explain the fact by believing that the patient really perceives but is not conscious of the perception. The same phenomenon is found in tests of *cutaneous sensibility*. It frequently happens that the patient, to whom, in the test, one announces each prick by "now," and who, according to the place touched, answers "no" and "yes," reacts with "no" to every unperceived touch, even when the announcing signal is omitted. If this phenomenon were not met with very frequently in patients who are beyond suspicion as far as *exaggeration or simulation* is concerned, one would conclude rightly that the patient was simulating. This could not possibly be true, and can as a rule, be excluded in the majority of cases. The patient distinguishes, without clear consciousness of the fact, between the pricks that are felt and those that are not felt, and designates the different feeling on the anæsthetic side as *not felt*.

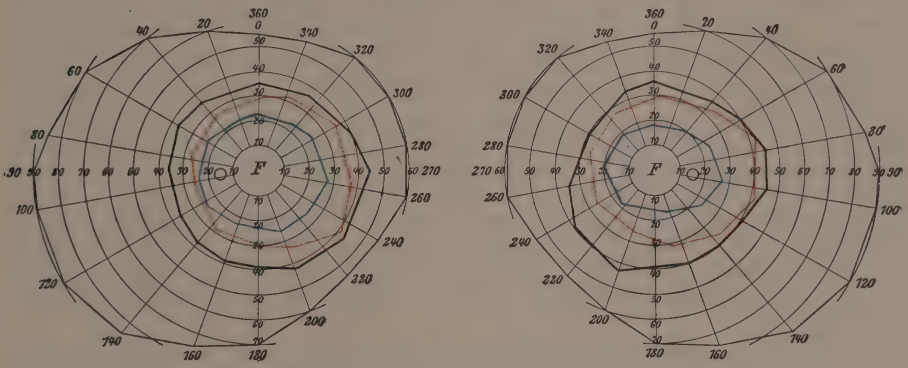


Fig. 210.—Typical bilateral concentric contraction of the visual field for all colors.

Hysteric *deafness*, too, has been repeatedly observed, mostly bilateral. Perhaps the cases in which children apparently fail to hear for years, so that they are often considered deaf-mute, or idiotic, belong under this head. They do not react to rather loud noises and calls, not even by shrinking; the sounds they emit do not, however, correspond to the rough tones of deaf-mutes. In later years, hearing and speech appear spontaneously. In one case such a child occasioned great surprise by suddenly repeating a song.

Unilateral deafness, therefore, eludes observation and escapes even the attention of the patient, because we are apt to attach but slight importance to distinguishing with which ear we hear.

The senses of *smell* and *taste* may be wanting on one side, not always, however, in like degree for all qualities of smells and tastes. This phenomenon, also, is apt to attract the attention of the patient only when it occurs bilaterally. *Hyperæsthesias* of taste and smell are more frequent; especially in this respect, that odors that are scarcely noticeable are very unpleasant to the patient, and create strong sensations of displeasure.

Disturbances of the *sense* of *equilibrium* have been little investigated as yet. One finds very often an uncertainty of gait with feet placed one in front of the other, still more frequently, visible *tottering* when the *eyes are closed*. Very characteristic is the following test, which discloses the psychic genesis of the phenomenon and distinguishes it from the similar phenomenon in tabes or cerebellar disease. In a patient who, with his feet close together, totters as soon as he shuts his eyes, and who immediately afterwards with open eyes brings his middle fingers together gradually, as in the test for intention tremor, and, who repeats the same experiment with the eyes closed, the tottering almost always disappears. In this test the attention of the patient is turned to the arm movements, and the tottering, which otherwise would be present, disappears. It is true that even in tabes, occasionally, the uncertainty disappears when the attention is attracted elsewhere, but in this case the tottering with closed eyes is more noticeable than in the hysteric.

At times, in hysteria disturbances of equilibrium that appear suddenly remind one very forcibly of the attacks of vertigo in lesions of the labyrinth. This *pseudo-Ménière's* disease is distinguished from the real *Ménière's* disease by the absence of ear findings and by yielding to treatment. In one case I saw after a labyrinthine affection accompanied by all its characteristic symptoms, besides attacks of vertigo other attacks due purely to psychogenous causes.

Since in the thoughts of the hysterics their own *body* plays such an excessive rôle, it is not surprising that they repeatedly complain in touching tones of *physical infirmities* of all sorts. Some of these complaints are so frequent that they have been called, it may be unjustly, pathognomonic. To these belong *clavus* and *ovarialgia*.

By clavus is meant a piercing *headache*, which, as a rule, is located in the vertex of the *head*, and feels as if a nail were being hammered in; sometimes this pain appears more towards the side or the back of the head, or even in the temples. Nor is its character always that of a boring pain, but frequently assumes a burning feeling or even a dull, not exactly describable sensation. Or the patient complains of a feeling, as if an iron band were fastened about his head. Sometimes he localizes disagreeable sensations *within the skull.*

The *ovarialgia* corresponds, in position, more or less, to the seat of the ovaries, and therefore, in former times, was frequently thought to be associated with diseases of these organs. This place of pain gained special importance by *Charcot's* demonstration that typical hysterical convulsions might be excited by pressure upon this point, and by further pressure caused to disappear again. (*Charcot's spasmogenetic zones.*)

I myself have seen patients of *Charcot* who always wore a strong elastic pelottes, in order to prevent the appearance of convulsions due to pressure. Further observations have, it is true, established the frequency of sensitiveness to pain in this region; on the other hand, the pain can not be connected with *disease of the ovaries*, inasmuch as it is nearly always located somewhat above those organs and is found, too, in hysteric men. It is now generally recognized that both the appearance and the disappearance of convulsions upon pressure at *Charcot's* point, is the effect of suggestion.

Further *points of pain* are found beneath the *mammary glands*, and along individual *intercostal nerves*, over the *spinal processes* and other parts of the body (Fig. 209*e*). Spasms occasionally result after pressure over these points. Painfulness in the *coccyx* is very frequent. It may become so severe, that it constitutes the patient's chief complaint, preventing him from sitting down, and annoying him when lying down; formerly, therefore, they occasionally extirpated the coccyx, sometimes successfully as regards the pain, sometimes not. For in these cases, too, we are not dealing with organic conditions.

Too much caution can not be taken in judging the pains of hysterics. Pains typical of ulceration of the stomach, sensations of pain in the gall bladder, pains in the regions of the kidneys, and above all, of recent years, in the appendix, have frequently caused false diagnoses and superfluous operations. I should like to emphasize here, that extraordinary caution should be taken in the case of hysterics before assuming that fresh pains are always hysteric. It may readily be seen that they may sometimes be due to organic causes, though I believe that the opposite mistake, that of erroneously assuming an organic foundation, is more frequently made.

A strange symptom, remarkable in so far at least that it has not been clearly explained, is the so-called *globus hystericus*. This is the name of the feeling as if a ball or some hard object were lodged under the larynx or in the œsophagus, as if a hindrance had to be overcome there in swallowing, or, very frequently, a feeling as if a knot, a feather, a bubble, were rising and falling in the œsophagus. This unpleasant sensation may occasionally lead to a *fear of swallowing*, so that the patients shun all food, and may even absolutely refuse nourishment.

The above enumeration does not include all the host of painful places and physical ailments. But to adduce all the possibilities, or only the more frequent ones singly, would mean counting up every organ, every region of the body.

Vaso-motor and Trophic Disturbances. Among the most debated questions of hysteria is this—whether *hysteric fever* really exists. I have never met a case that did not admit of doubt. The cases in which the temperature has been taken by the physician in such a manner as to exclude every deception through rubbing of the thermometer, and which hence can be referred only to hysteria, are very rare, even in the literature. Therefore many authors refuse to accept the fact that hysteric fever exists. It is never absolutely impossible that in spite of most careful physical investigation by a good observer a *physical illness* occasioning the rise in temperature may be overlooked. Still it is questionable whether one can deny the possibility of purely hysteric fever on general principles, if one considers, how very sensitive to suggestive and autosuggestive influences the *vaso-motor system* is in hysterical individuals.

A very great number of hysterics show, after simple *touching of their skin* with the tip of the finger, a deep and rather *persistent red.* After a somewhat rougher stimulation of the skin, as stroking it with the handle of the percussion hammer, very often this form is seen: to the right and left of an entirely anæmic stripe, which may be slightly raised, a dark red *discoloration* appears. In others, a more or less marked *wheal formation* appears frequently without a touch on the skin but merely because of ideas, especially those that arouse disgust.

Nearly always paralyzed or contracted limbs show a bluish-red coloration. This discoloration, designated also as *blue edema* (œdème bleu), and mostly associated with a swelling of the parts of the body affected, occurs also without any recognizable cause, and can, as well as the anæmic variety known as *white edema* (œdeme blanc) remain for weeks or even months. Very often these *swellings* are excessively painful and annoying, not seldom confined to very small and symmetrical places on the body.

Whether *trophic disturbances* actually appear in the form of *ulcers* is not definitely established. By far the larger part are certainly artificially produced by the patients. The desire to deceive and the virtuosity of the deceit is so strong in hysterics that one can not be too careful in respect to all such remarkable phenomena. But after exclusion of all doubtful cases there still remain sufficient data to prove to us that in hysterics the vessels react with unusual ease and liveliness to stimuli of all kinds, whence it may be, that in very rare instances, local anæmia leads to the formation of ulcers, or that a rise in temperature may be caused by vaso-motor influences. The expression "*hysteric rise in temperature*" would be more apposite than that of hysteric fever, because the usual concomitants of fever, acceleration of the pulse, drying of the tongue, stupor, increased concentration of the urine, are generally absent.

Attacks of pulse acceleration are not a very unusual symptom; occasionally in excitement, the pulse rate may be raised to an extraordinary height. *Pulse retardation*, on the contrary, though observed a number of times is a rare occurrence. Pressure on painful places, as a rule, gives rise to acceleration of the pulse which otherwise remains undisturbed qualitatively. Rapid movements produce the same effect.

In connection with the vaso-motor we may speak here also of the *secretory disturbances*. Among these unilateral *sweating* (due to excitement), profuse *watery diarrhœa*, without any other sign of digestive disturbance, excessive running of the nose (I observed it once for weeks, at exactly the same time of the day, lasting always an hour), are the most frequent. Also a long persisting polyuria, the hysteric nature of which may be proved by the effectiveness of treatment, is no unusual symptom, whereas one may be justifiably skeptical as regards cases of *anuria*. In a case known to me a patient had for some time simulated anuria, by applying for the headache wet cloths. which she had soaked with her urine.

Disturbances of Movement. Paralyses and *Contractures. Paralyses* may appear suddenly, apparently without any external cause (in one of my cases, a patient awoke with paresis of the arm after dreaming that he had been injured in a scuffle), but are mostly connected with a fright, an injury, or some such readily comprehensible cause. Or they may develop gradually, appearing first as a feeling of weakness, and then gradually developing into total paralysis.

It is difficult to estimate how frequently paralyses occur in hysteria; but seemingly strange local differences appear. Thus *Briquet* and *Voss* find paralyses in about 25% of all hysterics, whereas, according to my own observation, which coincides with that of other, especially German authors, total paralyses, and even partial pareses occur far less frequently.

Paralyses of hysteria are either *flaccid*, or associated with *contractures*. They may be *unilateral* and thereby simulate cerebral hemiplegias, or they may be *paraplegic* or *monoplegic*, or confined to the hand or foot. In the majority of cases, one finds as concomitants of the paralyses, *senory disturbances* in the region of the sensory organs of the same side, and, less often, of the other side. The *reflexes* remain normal or show exaggeration on the paralyzed side, that can extend to foot clonus and patellar clonus. *Babinski's* reflex on the other hand has never been observed. The appearance of the paralyzed limbs may, even in protracted paralysis, remain absolutely unchanged. Occasionally the paralysis is combined with an edematous swelling or with a *swelling* having a feeling of greater tension than edema, and with *discoloration* of the limbs affected. Cases showing muscular atrophy of high degree without change of electrical excitability exist side by side with other cases in which there is no muscular atrophy. In hysteric paralysis the strong *contraction of the antagonists*, in an endeavor to raise the paralyzed limbs, is noticeable (Fig. 211).

Less often the *facial muscles*, *tongue*, and the *external muscles* of *the eye* and only very rarely the inner muscles of the eye are affected. But cases of loss of *pupillary reaction* (Pupillenstarre)—not of the reflex, however—have been described by reliable authorities.

The *internal organs*, also, show occasional paralyses. Among these, belongs, first of all, hysteric aphonia, appearing as absolute *dumbness* or in a form in which the patient can barely speak in a low, hoarse voice; only seldom has total inability to speak been noticed. Also hysteric *stuttering*, which occasionally appears only in the presence of certain persons, or when certain letters are concerned, is not infrequent. Generally, it is a merely psychic disturbance, but one notices associated with it *spasmodic* movements of the tongue, lips, and larynx.

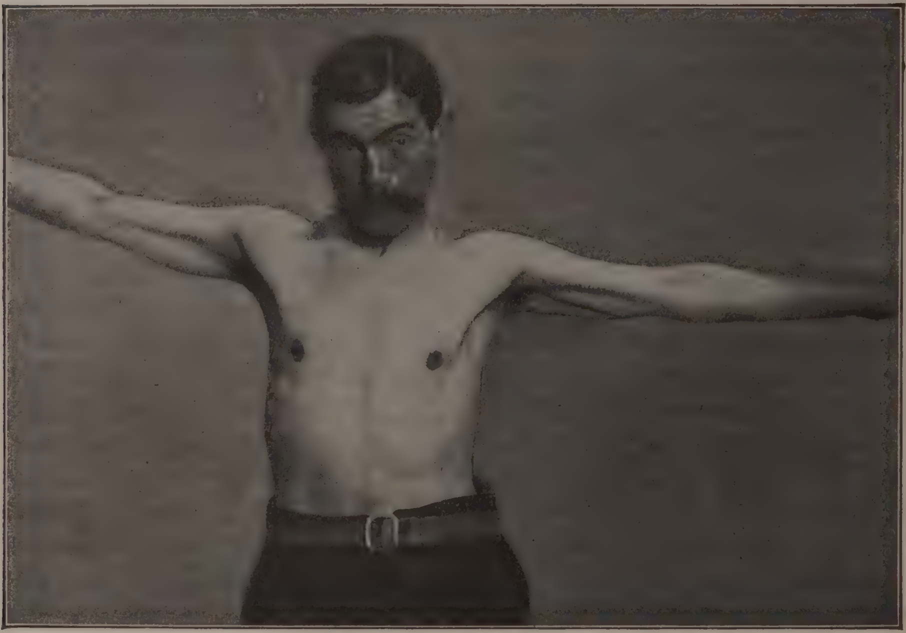


FIG. 211.—Paresis of the left arm with severe emaciation. Cramp in the musc. coracobrachialis. Photograph taken during the attempt to keep both arms stretched in the same height.

Hysteric *diarrhœa*, in so far as it must not be interpreted as a secretory anomaly, might stand in some connection with paralysis of the intestines, and possibly of the sphincter. I have certainly observed in several cases serious *bladder paralysis*, which *Oppenheim* considers very rare. It is not so rare, however, if one is inclined, as I am, so to classify *nocturnal incontinence* that has persisted for many years. This opinion seems to be justified by the results of therapeutic measures. But, excluding these, I know several cases of hysteric bladder paralysis, which uniformly displayed the peculiarity of appearing regularly only with definite psychic presuppositions, and on very definite occasions. Psychogenetic *urinal pressure*, which leads only to painful sensations, not to direct passage of the urine, is very frequent.

Astasia-abasia, a disease first described by *Blocq*, deserves special consideration. Precisely in this form the psychic genesis may occasionally be very nicely recognized. The patient is so convinced of his inability to walk and to stand, that, when put upon his feet, he allows himself to sink to the ground, whereas, he can move his legs perfectly well in bed. *Moebius* emphasized as a characteristic, that the patient in lying and sitting, can move his legs freely, whereas standing and walking are wholly impossible to him. I should not, like *Cramer*, distinguish cases described under the name

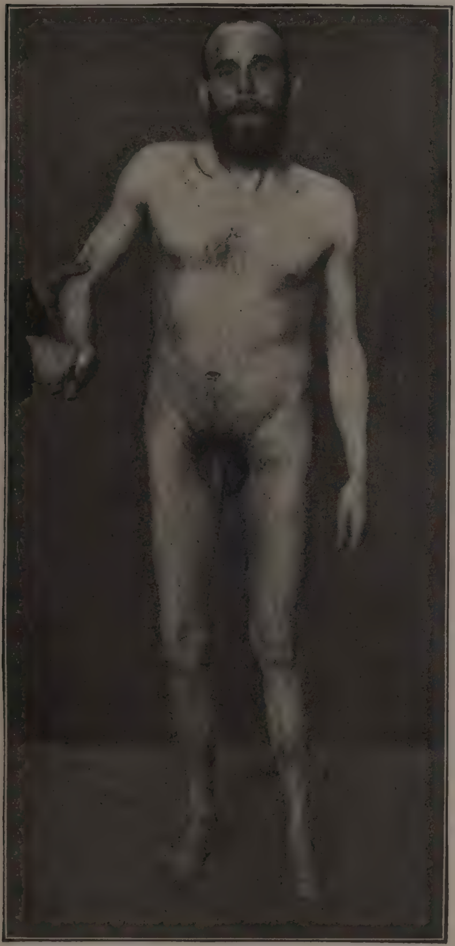


FIG. 212.—Hysteric dysbasia. Starting to walk; strong contraction of the toes.

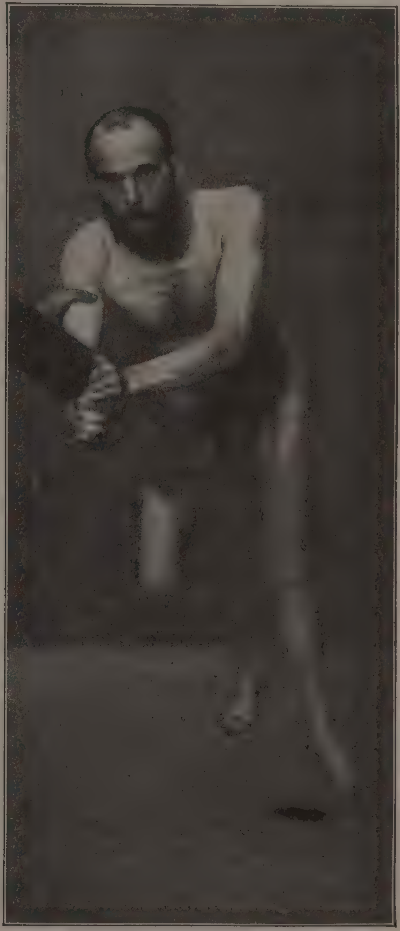


FIG. 213.—Hysteric dysbasia. The same patient while walking fast.

of *Stasobasophobia* (*Mingazzini*) from Abasia-astasia, since, in the individual case, the psychic process that gives rise to the phenomenon, is not sufficiently proved to make us consider *anxiety* as excluded.

More frequently than total inability to stand and to walk, occur *disturbances in walking.* Mostly associated with all sorts of *spastic* phenomena (*Nonne's pseudo-spastic paresis with tremor*) they give a peculiar character to the walk, which, from the beginning, bears but little resemblance to the walking disturbances in diseases of the spinal cord. If one observes the patients more carefully, one sees how they make walking almost impossible by purposeless tension upon various muscles. In Figs. 212 and 213, these phenomena are very nicely shown. The patient, who supposedly could not walk one step, can keep his balance, even in difficult postures—in Fig. 213, he is being led, at a quick pace, through a large hall.

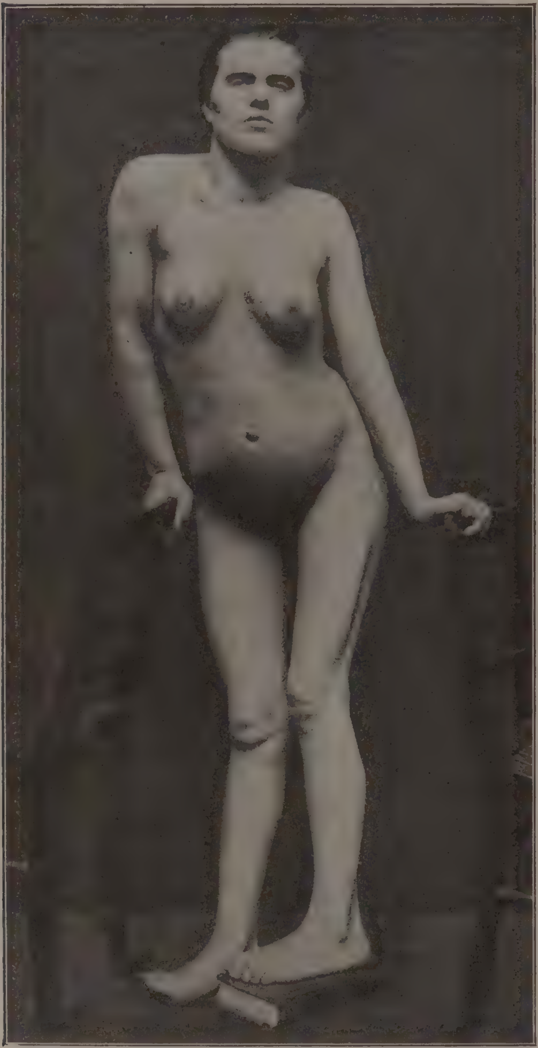


FIG. 214.—Hysteric contracture of the left hip. Cure by suggestion. (*From Schoenborn Krieger.*)

Tension of the muscles accompanies hysteric paralyses almost regularly. Besides they often appear, even independent of each paresis, in such a fashion that the French have even spoken of a *contracture diathesis.* However, the contractures are certainly not so frequent in Germany, that this designation is justifiable. They appear as *permanent contractures*, in which they fix a limb or joint in such a way that every attempt to flex it, or, in case of flexed contracture, to extend it, fails. One notices very frequently that in every passive change of position, the already strong tension of the muscles is immediately increased. The permanent forms of contracture may remain for years unchanged. Sometimes, if not always, the contractures persist even during *sleep;* in *chloroform narcosis*, they relax, or, at least, may be relaxed passively.

I think, as opposed to *Binswanger, that psychic ideas* play an important rôle in the origin of the contractures. This is proved by the possibility of replacing contractures, in hypnosis, by means of the *magnet* or Franklinisation, or by the application of metal, which *transfers* the contracture to the other side. I saw once an extension contracture of the leg, the result of a flexion contracture that had been extended in narcosis, and fixed in extension for some days in a plaster cast.

Besides all of the limbs which are usually affected in toto (Fig. 214), other muscles are affected by long protracted contractures, which are usually accompanied by pain. In comparison with those in the extremities contractures in the region of the facial muscles are of rare occurrence. Occasionally contractures develop in the region of certain groups of muscles which fix a diseased joint; but the muscular spasm persists long after the pain in the joint has disappeared.

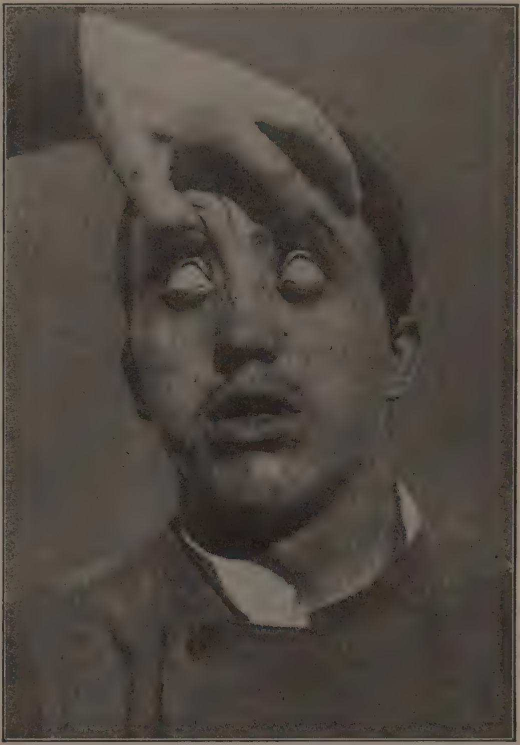
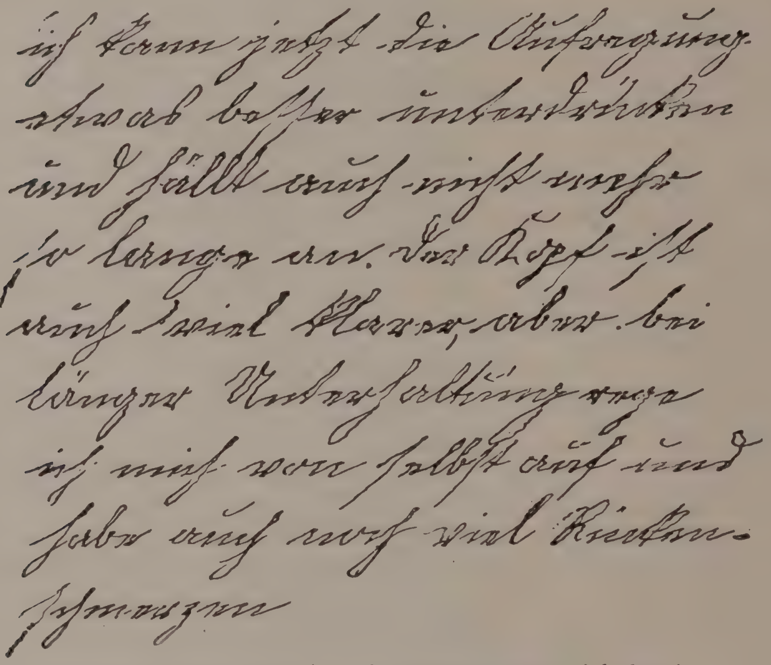


FIG. 215.—Hysteric convergence spasm of unusual intensity. (*From Schoenborn and Krieger.*)

Besides these long continuing contractures, there are others that appear *intermittently*, at times in a form in which pressure on certain *hysterogenous* zones causes certain muscles to contract. At other times contractions develop in the course of definite movements. In Fig. 211, is shown contraction of the coracobrachialis in an attempt to maintain the paretic arm in the same position, as that on the unparalyzed side. The tension upon the muscles continues for a short time even after the arm has been lowered.

In cases of permanent, as of intermittent contractures, one will rarely fail to find *disturbances* in the *cutaneous sensations*, whether they be in the form of anæsthesia or hyperæsthesia. I indeed agree with *Binswanger*, that the *pain* in the overstretched muscles is scarcely ever absent. The contractures deserve notice too for the reason that the contraction of the antagonists occasionally simulates paresis. At any rate they belong to the most annoying phenomena in the clinical picture of hysteria.

Here, too, may be mentioned *hysteric tremor*. According to my experience, a typical hysteric tremor does not exist. We recognize as hysterical cases of extraordinarily slight, almost unnoticeable, tremor, which becomes intensified, only in intense emotional excitement, as well as cases, in which occur quite coarse, almost thump-like twitchings, which sometimes have *something about them that seems purposive.* As a rule, the trembling diminishes and may entirely disappear, if the patient's attention is *turned elsewhere.* But in others it persists even then. Hence is explained why in some individuals whose trembling is very marked, written script, which as a rule reproduces the trembling movements very nicely, is absolutely undisturbed, whereas in others (Fig. 216) the trembling may be readily recognized.



ich kann jetzt die Aufregung
etwas besser unterdrücken
und hält auch nicht mehr
so lange an. Der Kopf ist
auch viel klarer, aber bei
längerer Unterhaltung rege
ich mich von selbst auf und
habe auch noch viel Rücken-
schmerzen

Fig. 216.—Hysteric tremor (very characteristic contents of the letter).

The Hysteric Attacks. The classical works of the school of *Charcot*, especially *Richer's*, have, without doubt put an end to the stage of vague confusion as to the meaning and forms of *hysteric spasms.* It is surprising that these typical attacks of grand hysteria (*grande hystérie*) occur far less frequently in other countries. Today it may be considered as an established fact that nothing is so contagious as an hysteric attack. And if such attacks increase in the house of a patient, or in a hospital, the cause is, that too much *attention* has been paid to the attacks.

The Salpetrière, the place in which *Charcot* and his scholars did their work, not only afforded a haven for the assembling of the serious cases of hysteria, but also afforded opportunity for the patients to learn from one another. This is explained that nowhere else has so large a number of classical attacks been observed, and that since we have learned to ignore them more, their number and development have decreased considerably. At any rate, *Binswanger's* statistics as to the percentage of hysterics with attacks fall far behind those given by the French, and those given by Voss for St. Petersburg. I myself, in spite of the great number of hysterics I have treated, have none too frequently observed individuals with serious attacks of the kind described by *Richer*, or even of the rudimentary types.

The typical hysteric attack proceeds somewhat in this manner: *Anger*, grief, terror, or even joy; in some patients pressure on one of the *spasmogenous* points of pain, give rise to the attack, which is generally preceded by *prodromes*, consisting of general discomfort, sense of pressure on the head, sensitiveness in the region of the stomach, a feeling of being inflated, globus, yawning, and when the patient's mental attitude is that of anxiety, occasionally of a single hallucination. Then the patient falls down, more or less suddenly. Since *consciousness*, for the most part, does not vanish any too rapidly, the patients do not, as in epilepsy, fall down, as if struck by lightning, but can still look around for a chair, or a bed, or can let themselves sink gradually to the floor. But I wish to lay special stress upon the fact, that we may observe the same phenomena, occasionally, in epileptics. In this long and *gradual sinking* down *injuries* virtually never occur. I can recollect observing only twice an injury worthy of the name: once immediately after I had pointed out to the patient the absence of any injury to herself, in spite of her numerous falls, as a proof of the non-epileptic nature of the attacks.

Now begins the *stage of convulsions*, *Richer's période épileptique.* They resemble very frequently the *disordered twitchings*, the wild irregular motions of the limbs in an epileptic fit, so that differential diagnosis is extremely difficult and can be established only from the concomitant manifestations of the whole disease picture, from the course of the attack and the effects of therapy. It is true, that for the most part in hysteria the twitchings are rather of the nature of a *co-ordinated movement;* the patients pound, hammer, push and tread, often quite rhythmically. The movements do not, as a rule, last long, often *tonic rigidity* of the entire body predominates.

The *color of the face* remains unchanged or becomes pale; one rarely sees an intense cyanotic discoloration of the face and the body. If one attempts to open the eyes (at times they remain wide open), the bulbus looks upwards, so that it is not easy to test the *pupillary reaction.* It seems rarely to be absent.

In one case, I happened to investigate, by chance, the pupillary reaction and was astonished to find a loss of pupillary reflexes. Immediately afterwards the patient fell into a typical hysterical attack.

Biting of the tongue is very rare, in spite of serious convulsions, and then usually it is merely superficial. On the other hand, I have upon different occasions seen involuntary *passage* of the *urine* in undoubted hysterical attacks. The *reflexes* are, as a rule, unchanged, at times increased to ankle clonus. *Sensation* is abolished; but in this, as in other stages, the characteristic observation is occasionally made, that an energetic pin prick or sprinkling with water is followed by a distinct reaction, for the first time only, but not again.

After a not always clearly defined *resting period*, in which the patient lies, wholly relaxed and exhausted, the second stage begins, *Richer's period of contorsions and of grand movements* (*periode des contorsions et des grands mouvements*). The patient begins to turn round and round, throws his legs into the air, does gymnastic acts, or bores his head into a cushion, a characteristic movement, which being extremely frequent, occurs even when a *semicircle* is not formed. In the typical "*arc de cercle*" the whole body rests on the heels and the head (Fig. 217). The *variety of the positions* assumed by the patient is very great, and one has often the impression of witnessing a professional *contortionist*, even in the case of persons usually awkward. The name *clownism*, which *Charcot* gave to this phase of the attack, characterizes the peculiar postures very well.

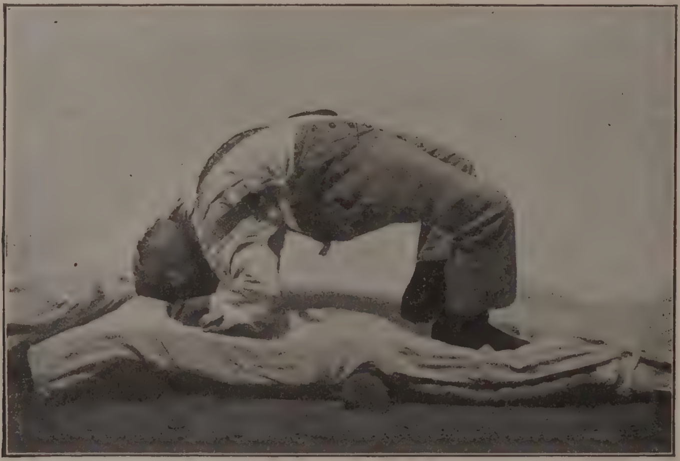


FIG. 217.—Hysteric arc de cercle in a 9 years old boy. (*From Schoenborn and Krieger.*)

Gradually they assume a *voluntary, attitudinizing*-like character; movements of greeting and threatening, bows, dancing steps form a transition stage to the period of *passionate attitudes* (attitudes passionelles). I see no cause and no possibility of separating this from the *period of delirium* (*periode de delire*) which *Richer* describes as a fourth phase. The patient jumps up, his attitudes, his features make it possible to recognize that he is a prey to *strong emotional excitement.* He plays the actor—in this stage, unconsciously —carries on a long speech with pathetic intonation and lively gestures, flees from a snake, from a wild animal, seeks to escape a conflagration, defends himself violently against accusations, which he seems to hear. Or he acts like a child, plays with a doll, gathers flowers, chases butterflies, climbs upon tables and chairs. Or he falls into an *ecstatic trance*, sees visions from heaven, to which he prays at great length, with deep feeling. It must be understood that this phase, which may last for hours, in which the patient may move and speak with an ability and freedom often impossible to him at other times, creates a striking *impression of an individual perfectly normal*, which easily deceives the ignorant as to the worth of utterances, prophecies, reminiscences, made at such times, and frequently gives rise to the idea, that the patient is *possessed* or is favored by God.

The attack closes with a *sleep*, which is only exceptionally of long duration, or goes straight over into perfect *sanity*. Only where the convulsive stage has been of long duration is the *exhaustion* great. At other times the patients feel surprisingly well, considering the long duration of the attacks.

Opposed to these grand attacks are the so-called *rudimentary* attacks, in which one or more phases are omitted, or but briefly suggested. So one occasionally sees the patient suddenly bore his head into the pillow, stare in front of him for a short time, and then recover. Or convulsions or tonic rigidity appear, ending in a short time, without the addition of another stage. Or, only theatrical scenes occur, and there are no convulsions. Occasionally the attack is confined to a shorter or longer period of dimming of the consciousness, wherein the incongruousness between the apparent possession of his faculties and the inability of the patient to answer simple questions, is the most conspicuous symptom (*Ganser's* twilight state).

At any rate, experience has proved that exact division into three or four phases is impossible, that the various conditions go over into one another, and that the completed development of the attack in the way described is very rare.

Very strange are the conditions designated as "*lethargy*" in which the patients suddenly fall asleep and under certain circumstances do not awaken for days. Such states have also been called *autohypnotic*, since they occasionally appear in patients who previously have been frequently hypnotized. But they occur far more frequently in patients, who have never in their lives been hypnotized. The coessentiality lies, doubtless, in the *suggestion* of the idea in the patients, arising from unknown causes, that they must sleep. The patient has only a fragmentary *remembrance* of such a state just as after attacks of grand hysteria the patient knows only part, or nothing at all of what has happened.

Whether one should enumerate here all the other attacks, seems to me doubtful. For instance, in hysterics, long *spells* of *coughing, sneezing, or yawning* occur, or the patient experiences a disagreeable *need to nod*, or at a sudden noise, the patient begins to *dance rhythmically*. These attacks are distinguished from those described above, in that they are never accompanied by loss or *dimming of consciousness*. The same holds true of *laughing* and *crying spells*. *Crying spells* are nothing but nervously exaggerated emotional expressions, which the patient is unable to control. On the other hand, laughing spells have frequently something forced about them, in so far as they do not correspond to the mood of the patient, but rather appear as a sort of contrast phenomenon in absolutely unsuitable situations.

Hysteric psychoses I am inclined to exclude from consideration. It would take me too far should I attempt to establish my conception, that we are not justified in separating hysteric insanity, as a disease *entity*. We know besides the already mentioned brief conditions of mental confusion, described by *Ganser*, other hysteric twilight states; but these resemble very closely the disease picture of grand hysteria. We know, furthermore, of the intermingling of hysteric traits in many diseases, as, for instance, in *manic depressive* insanity, easily comprehended because of their close relationship and their development on a substratum of endogenous predisposition. But the separation from forms of mental disturbance, which only *externally* show a certain resemblance in various symptoms, would be out of place here.

Differential Diagnosis.—Hysteria may *simulate* all forms of disease pictures with such a degree of perfection that it is at times difficult for the most experienced to avoid errors. The surprisingly genuine *imitation* of complicated forms of diseases becomes more pronounced, the more the patients are given an opportunity, to learn from other patients in hospitals, or great cultural centers. It is not a matter of *conscious imitation*, at least not as a rule.

The point of view, that the patient in all probability had no chance to see an organic disease, helps diagnosis sometimes, but by no means always. I saw, for instance, a diagnosis of *bulbar paralysis*, with typical bulbar disturbances in speech and swallowing, made by several good nerve specialists, in the case of a girl, who came from a distant village, and could certainly never have seen bulbar paralysis. The phenomena disappeared without any treatment, immediately after she entered the hospital. Years after, paraplegic phenomena appeared, which also disappeared.

But one can err also in the other direction and *too frequently* diagnose hysteria. I shall mention only a few of the many cases that I have seen. *An itching of the skin*, thought, because of other nervous troubles, by skin and nerve specialists (among the latter I reckon representatives of internal medicine) to be psychogenous, was the consequence of faulty functioning of the gall bladder. They had neglected to test the urine. A nervous child, with *fits*, was supposed to be hysteric, until the careful investigation of the vocabulary clearly disclosed asymbolia and, hence, the organic foundation of the disease. By an examination of the fundi oculi I have repeatedly found *cerebral tumors* and *multiple scleroses*, where hysteria had previously been diagnosed. *Facial neuralgias* developed as suppurations in the antrum of Highmore, general *nervous debility* as diabetes.

Such *false diagnoses*, which have probably occurred to everybody, and some of which everyone has made, may only be avoided, if nothing is left out from a thorough examination, from the examination *of the eye-grounds* to the *testing of the urine*. In this, I include not only the physical functions, which the physician would of course have to investigate, but should urgently advise that much attention be turned to the *psychic condition*. Then *ulcers* of the *stomach* will not be diagnosed, where an exact test of sensibility would have disclosed the fact that besides the hypersensitiveness in the region of the stomach, other hyper- or hypo-sensitive places exist, or *appendicitis*, where a simple ovaralgia is concerned.

The diagnosis of hysteria must necessarily begin by establishing the *constitutional condition*. This must always be remembered. For we find, on one hand, all sorts of *psychogenous traits* in non-hysterics, and these very frequently as concomitants of serious organic diseases; and, on the other hand, the *stigmata* are frequently absent, or so concealed, that only a laborious investigation—such a one, for example, that in the sensibility test neglects no part of the body, and examines all sensory qualities—may disclose them.

In general, it is in the interest of the patients, if in doubtful cases, the physician inclines to the more *serious* views of the *disease*, because he then runs less risk of missing something essential in the *treatment*. This, however, does not hold good for *hysteria*. It can only be *intensified*, when too great attention is turned to the individual phenomena, and the many cases of false *operations* performed on hysterics, because of false diagnoses, are the best proof that the physician can not be careful enough with hysteria.

He must be cautious of *mistakes* in two directions. In one, that he must not so far be led astray by the theatrical manner of the complaints or the superficiality of the troubles as to *overlook organic diseases*, in the other, that he may avoid the assumption of the existence of organic diseases, where there is nothing but *psychogenous phenomena*. This must never be overlooked: individual symptoms that we generally call hysteric, are per se, no proof of hysteria, and, on the contrary, hysteria can not be excluded, where we do not find stigmata immediately springing up to meet our gaze.

If one wished to state, which diagnostic difficulties need particular attention, it would be necessary to discuss virtually all diseases, one after another. Therefore I shall confine myself only to the most frequent. The most important is its demarcation from *epilepsy*. I incline, contrary to *Cramer*, to deny the existence of so-called *hystero-epilepsy*. Naturally it can not be denied that a hysteric, possibly after an injury to the head, might have typical epileptic seizures, nor that an epileptic may exhibit also all sorts of hysteric manifestations. But the two diseases are so different, that the conception of hystero-epilepsy necessarily leads to a weakening of the conception of each disease and to errors.

I can not admit, that there are convulsive attacks, which one could, with *Oppenheim*, fitly call *intermediary*, standing between epilepsy and hysteria. I am willing to admit that even for an expert it is often most difficult to assign the individual attack to one or the other disease. But, as a matter of fact, the attack is only the symptom of the disease. The disease epilepsy does not consist only in convulsive attacks, but shows attacks of other sorts and a peculiar moulding of character; on the other hand, hysteria, too, has besides the attacks, its own peculiarities, which must be investigated, if one wishes to make a correct diagnosis.

In the individual *attack* one may base a diagnosis of epilepsy on the presence of pupil immobility, biting of the tongue, and other such injuries, on the special kind of motions, on the passing of urine and feces and on the peculiar stage following the convulsive attack. The epileptic attack is unfavorably influenced by alcohol, the hysteric is mostly released by emotional stimuli. The epileptic falls into convulsions everywhere, even where, as on the street, the bustling crowd directly *threatens his life*. The hysteric attack occurs mostly where there is a limited circle of spectators, scarcely ever when the patient is unobserved. The epileptic attack is *uninfluenced* by external interference; the hysteric attack can, under certain conditions, be made to disappear immediately.

I emphasize again here, that no isolated phenomenon should be used, alone, for or against a diagnosis, but all the symptoms should be considered. And to these symptoms, besides the attack, belong the following: the character of the patient, his conduct, disturbances in sensibility previous manifestations and stigmata. If all this is taken into account, a conclusive differential diagnosis of epilepsy from hysteria may be made in every case.

The differentiation from *organic brain and spinal cord diseases* may occasion great difficulty, and especially that from multiple sclerosis. Where there is a spastic-paretic gait, permanent nystagmus, clearly defined intention tremor, the diagnosis is not difficult. But opinions differ even about the significance of *ankle clonus*. Indeed, even the *Babinski toe reflex* is supposed to have been observed in hysteria. I have never seen this, but I have seen unmistakable ankle clonus, which therefore, with *Oppenheim*, in opposition to *Jolly*, I must assert is worthless as a pathognomonic symptom. The less defined the manifestations, the more difficult is the differential diagnosis. *Temporal pallor* of the pupils settles the diagnosis, though I believe it does not appear so frequently as an early symptom of multiple sclerosis, as *E. Müller* has stated.

Organic brain diseases, too, have often caused mistakes—especially *tumors of the brain* without clearly defined localized phenomena. The headache is duller in brain tumor, the stupor is greater, than in hysteria. Where vomiting and changes in the pulse indicate pressure phenomena, changes in the *background of the eye* are seldom absent. But one should never forget that psychogenous symptoms have been frequently observed in brain tumors.

Of other nervous diseases, I shall call attention only to the hysterical *neuralgias.* It is often very difficult to distinguish between them and other sorts of neuralgias. At times assistance is given by ascertaining, that the pains go beyond the realm of the nerve attacked, and spread in other directions, at times, that in the region of the neuralgias, *anæsthesias*, and more frequently, *hyperæsthesias* of the skin are found, occasionally also, the *symmetrical involvement* of the nerves of both sides, or the symmetrical situation of a comparatively large number of painful points.

In the realm of physical diseases, the most important difficulties in differential diagnosis arise from *localized pains.* They can, apparently, be localized so characteristically in certain places, as to simulate, for instance, *ulcers in the stomach.* If to the ulcer symptoms are added loss of appetite, anæmia, and vomiting, which may even contain blood, the differential diagnosis becomes well nigh impossible. Also *gall-stone colics*, *appendicitis*, *inflammatory processes of the genital organs*, *inflammation of the joints*, occasionally with synchronous fixation of the joint by contractures, may easily be assumed, where nothing exists other than hysteria. In these cases, too, where physical diseases are to be distinguished from hysteria, one can not be too careful in the differentiation.

Prognosis.—The *prognosis* varies according as one considers *the removal of individual troubles or that of the entire disease.* One may say that every single symptom may be made to disappear, even though the obstinacy of some manifestations may exhaust the patience of the physician. One ought to hesitate more in making an assertion of incurability in hysteria or hysteric symptoms, than in any other disease. What fails with the deliberately aimed treatment of the physician, at times succeeds easily at the hands of a *quack.* Not in vain are hysterics, and also neurasthenics, the most faithful adherents of all sorts of quacks, and the most grateful subjects of treatment with blue electricity and voltaic crosses, with Kuhne friction sitz-baths and mud packings. They are the first to become enthusiastic over the healing methods of Christian science and ocular diagnosis. It is faith that cures (c'est la foi qui guerit) said *Charcot* of the methods for treating hysteria.

And if the belief in the effectiveness of any mystic method has cured them of their troubles—troubles that mocked the skill of the physician—the patient will always be inclined to see therein the incompetency of the physician.

The **prognosis** of the disease in its *entirety* must be quite differently regarded. Hysteria, in my conception, represents a constitutional predisposition. A patient *is* hysteric, he does not *become* hysteric; and he *remains* hysteric, even if all the manifestations remain in the background temporarily or even for years. Therefore, one may always expect new symptoms, and, in the treatment must aim to teach the patient how he may defend himself against the reappearance of the symptoms. One cannot begin too *soon* with the treatment in hysteria. Just because *educational factors* are of such great importance, the course of an untreated hysteria is quite different and far less favorable than that of one properly treated.

On the whole, there is generally no *danger of loss of life*. The significance of so-called *acute, fatal hysteria*, is quite unknown. The few cases described heretofore, are mostly of very questionable nature and do not exclude with sufficient certainty, the possibility of organic diseases of the central nervous system. As a result of persistent vomiting, profuse diarrhœa, and complete loss of appetite, the patient may, possibly, go to the verge of death, but whether beyond that, is doubtful. At any rate, in such a condition, a physical illness, possibly not dangerous in itself, may be a serious menace to the life of the patient.

Finally a word must be said regarding hysteric attempts to *injure* one's self and to commit suicide. They are not so harmless, as is generally assumed. The swallowing of glass and stones, the sticking of pins into the skin, have frequently caused fatal perforations. The suicidal attempts of hysterics are carried out in one of two ways: 1. The patient, to produce an effect, ostensibly attempts a suicide, that was never seriously intended. An unfortunate chance may bring about an unexpected end. Thus I saw a jump from a window on the ground floor end fatally because a nurse attempted to restrain the patient. In this way the patient lost her balance and suffered a fracture of the skull, that eventually killed her.

2. Patients commit *suicide* in emotional states that are seriously intended and that end seriously. In these cases, it is not a matter of long pre-arranged preparation; the patient is carried away by his *emotions*, and loses that calm deliberation, which might have shown him, at other times, that the cause was far too insignificant for suicide.

2. ACUTE NERVOUS EXHAUSTION

Definition.—We know from experimental physiology that the work of the muscle, by aggregation of disintegration products, leads to the diminution of its functional capacity, that we call *fatigue*. If the work is continued, without giving the muscle time to recover and without the removal of the disintegration products by metabolic processes, the strength supply of the muscle is used up, exhausted. A sharp line between fatigue and exhaustion cannot be drawn even in the laboratory in working on muscle preparations, certainly not in life, where psychic exertion is an additional injurious factor to the physical, the emotions to the work. Therefore we shall have to give up an attempt to draw sharp distinctions between fatigue and exhaustion. They shade off into each other; if the exhausting influences follow in too close succession, and the periods of recuperation are too short, a demand in itself slight, may accelerate the collapse of their forces. From a practical standpoint, then, a superficial distinction between the conceptions of fatigue and exhaustion suffices, so that by fatigue we mean a *transitory diminution* of functional capacity, which may easily be compensated for, whereas in serious cases of exhaustion, called forth mainly by *protracted fatigue*, it may take a long time until the former tonic power is restored.

The stronger the body, the more clearly the clinical picture of fatigue appears, the more severe and continuous the damaging influences, the more severe is the exhaustion of the forces. But the sound organism remains, even in that case, free from all the traits, that we meet on the ground of congenital psychopathic constitutions. Thus it was only a natural development in the conception of *neurasthenia*, that two originally combined diseases were again separated. *Kraepelin* differentiated the disease contracted and developed on *non-degenerative* foundations, under the name of *chronic nervous exhaustion* (*Cramer's* neurasthenia) from nervousness, which Cramer in order to call attention to the predominance of *endogenous* phenomena, names "endogenous nervousness." Inasmuch as nervous exhaustion may develop both acutely and subacutely, and the designation "chronic" may easily give rise to the misunderstanding, that a permanent condition is concerned, I should prefer for this form the name "acute nervous exhaustion." The adjective acute is intended to designate the development of the disease on a sound foundation, the remediable as opposed to the constitutional condition. The term "neurasthenia," once adopted, I should retain, on the other hand, for endogenous conditions. It seems to me thoroughly practicable, especially if we insist that the symptoms of neurasthenia are consequences of *constitutional nervous exhaustibility* and therefore always *endogenous*.

In last analysis, however, both forms are really interchangeable; the more predisposed the individual is to exhaustion, the more quickly acute exhaustion must appear, only that in such cases, we find phenomena conditioned by the inborn predisposition, besides those of the acute exhaustion.

Etiology.—Every *overexertion* may lead to exhaustion, if the strength on hand is used, without obtaining sufficient replenishment. Especially after serious *physical* illnesses do conditions of great exhaustion frequently follow. The experience, that not all show the same effects, that particularly after infectious diseases, especially typhoid and influenza, the regaining of the old functional capacity is often delayed, makes us suspect that toxins are of some importance in the matter. But we find similar symptoms of exhaustion also in non-toxic diseases, as after serious obstetrical cases, great loss of blood, as well as after painful diseases which for a long time rob the patient of sleep.

Likewise acute nervous exhaustion develops after very severe physical and especially after severe mental exertion. One may even safely say that no cause is so often assigned as *overwork*. This word has an ill sound inasmuch as it has become a catch phrase, serving as a pretext for a superfluous and very frequently a dangerous effeminacy. But from these exaggerations one must not conclude that there is no such thing as overwork. Functional capacity in the physical as well as in the mental realm is subject to extraordinary individual differences, and a given piece of work may injure one person considerably and yet be far from occasioning another to go to the limit of his strength.

The demands of an active—and let us assume, in order to exclude the influence of emotional disturbances—prosperous business leave the energetic business man no time for rest; his meals are swallowed in haste, his days spent in feverish activity, his nights in travelling on a train, his evenings and Sundays are spent in working out new plans, until a breakdown in strength puts a stop to all this raging hurry. And likewise, the scholar, who faces the completion of a great work, the physician, whom a large practice and serious epidemics keep on the run night and day, the pupil, who, because he learns with difficulty, or because he is hindered by additional work (tutoring) or by all sorts of sports and fads, must utilize the hours of the night, in order to master the task assigned—all these are unavoidably hastening towards a breakdown, if *opportunity for recuperation* is not interspersed. If this is not had or if recuperation is insufficient, we see the signs of overexertion grow stronger and stronger, until one day both body and mind give out.

This time appears sooner, when intense *emotional disturbances* are combined with the increased demands on strength. Therefore cramming for examinations, under the continuous pressure of constantly anxious anticipation and wonder whether the time left will suffice for preparation, the business panics that endanger the merchant, the illness of members of his family, that robs the physician of his usual rest, are all extremely dangerous. The same holds true, to a certain degree, for cares outside of one's business or profession, because they deprive one of sleep and therefore destroy the best means of protection against over-exhaustion.

I attach no such great importance to another injury, namely *sexual excesses*, often emphasized as a cause of nervous exhaustion. I believe that the body is not weakened so much by the intensified and exaggerated demands upon the generative glands, as by the attendant circumstances—alcoholic excesses, loss of sleep, and the emotional disturbances, varying with the individual case (fear of discovery, of pregnancy, of infection). The greatest danger, as we shall have to explain in detail in our discussion of neurasthenia, is the irrational *fear*, perpetually stimulated by the excessive but very remunerative *production* of pamphlets and preventatives, of the dangers of sexual excesses, especially of *masturbation*. But here precisely is shown the difference between the healthy and the psychopathic individual. The mind of the healthy individual detects easily the *exaggeration* in all this literature, the attempted exploitation of tendency towards anxiety and of the pocket-book; he quickly overcomes—possibly with the physician's help—the fear that has been aroused, and thereby escapes the most dangerous part—the *emotional*. The nervously predisposed individual, however, is not easily calmed. He, on the contrary, draws from the pamphlets ever fresh material for hypochondriac self-observation and anxiety, and what physical exhaustion can not do, mental torture does, and his strength is sapped.

There remain then of the causes of acute nervous exhaustion only a few. They are always only *real over-exertions*, or *serious physical injuries* that we may regard as causes, and these are the more easily discernible in their effect, the healthier the individual naturally is.

Symptomatology.—The subsequent phenomena of overexertion express themselves chiefly by a great tendency to *fatigue*. The patient can not work long without interruption; he may feel quite fresh after a full night's sleep, but as soon as he begins to work, his strength gives out; he is turned aside and *distracted* by the slightest disturbance, begins to dream when he reads, soon discovers that for the last minutes he has been wholly inattentive, begins anew at the same point, without actually reaching his goal. The feeling of *fatigue* is so strong, that he does not trust himself to take up any serious activity although he may temporarily pull himself together on important occasions.

The patient thinks he notices, that his *memory* is apt to fail him, though no objective disturbance can be established. The *power to receive and retain a new idea* (*Merkfähigkeit*) is, however, actually *impaired*. Considering the close connection between the ability to impress on one's self new perceptions and facts, and concentration of attention, this is only too obvious. The patient thinking this failure to be weakness of memory, loses his poise and his firm confidence in his working ability. Then come complaints about the feeling of mental collapse, a feeling of emptiness in the head, inability to keep the thoughts together, anxious worry as to the future. The patient becomes sensitive, especially to noises, irritable, is easily angered, especially by trifles, which would formerly have found him indifferent.

Externally, this condition appears as slight *motor restlessness*. The patient is unable to sit still, or to remain in one room for any length of time. This restlessness is increased by psychic instability. He does not stick to what he is doing because all sorts of things occur to him, that must be attended to at once, and as soon as he begins something new, the scene is repeated.

This physical restlessness is very painful to him, in as much as it is also noticeable to outsiders, as well as his inclination to yawn. But if he attempts to sleep, no matter how tired he may be, he cannot fall asleep at once. The events of the day and his restlessness interfere. The desire for sleep, however, is extraordinarily great. But generally—and in this way especially these patients are distinguished from neurasthenics—the patient can sleep soundly at any time of the day or the night, once he has fallen asleep.

So long as the exhausting causes continue their work, sleep may elude the overtired. But as soon as he has fallen asleep, stupor (lethargy) appears to an extreme degree. It is a remarkable, if not a frequent phenomenon, that such exhausted people sleep in any position on any occasion.

The *physical condition* leaves much to be desired, though definite complaints may not be made. The patient feels worn out, powerless; the limbs become stiff, and give out upon any exertion; pains which are persistent stubbornly appear in the overworked muscles. The head is preoccupied, empty, feels as if it were surrounded by an iron band, in fact every movement of the head is painful. When reading slowly the letters dance before the eyes, and sight is hazy.

Sexual excitability is lowered, without disappearing. Every attempt at sexual intercourse increases the feeling of exhaustion. The *appetite* is small, but occasionally an almost insatiable *hunger* appears.

The *weight of the body* diminishes, especially in exhaustion following acute diseases and loss of blood: if the patient persists in his exhausting mode of life, he becomes thinner and thinner, in spite of the most ample nourishment. Finally, strength fails utterly.

Differential Diagnosis.—Acute nervous exhaustion is occasionally so closely related to constitutional neurasthenia, that no sharp division can be drawn. But, in the majority of cases, the former history, the absolute absence of any nervous trait in past life, and the proof of serious injury preceding the collapse, make the distinction possible.

The determination that really serious injuries preceded, is especially important because it is concerned in the most significant differential diagnostic difficulty, in distinguishing it from paresis. As a first symptom of paralysis we find, very frequently, undefined general nervous troubles of all sorts. Their appearance in middle age, especially between the ages of 35 and 45, is always suspicious, even when the nervous phenomenon is developed in an individual, who, before that period, has shown no signs of nervous disposition. Then one will have to use twice as much caution in judging whether the preceding injury was really serious enough to be a sufficient explanation for the loss of mental tonicity, and only then—presupposing the absence of physical phenomena—abandon the suspicion of paresis, if the cause seems really sufficient.

At a somewhat more advanced age, the differentiation from *arteriosclerotic* processes may present difficulties. The progressive diminution in functional capacity together with the appearance of the most varied nervous troubles, is common to both diseases. The aging body fails with reference to the same demands as does the over-exerted. But the prognosis in each case is absolutely different. It is a pity that the arteries, as felt externally, do not always give safe grounds for conclusions as to the state of the cerebral arteries. The increase in blood pressure, especially if persistent, and considerable is a more useful diagnostic factor as is also temporary albuminuria, neither of which belongs to a typical picture of nervous exhaustion. But in view of this difficulty in all cases it will be well to be very careful in the prognosis of acute nervous exhaustion in advanced age.

Earlier in life, too, especially about the period of development, conditions appear, often sailing under the false flag of nervousness or nervous exhaustion, whereas in reality, it is a matter of insidiously developing *hebephrenic states.* The difficulty of diagnosis is increased by the fact that patients may often still be able to carry out fairly well the daily occupations, to which they have become accustomed, and fail only when serious demands, especially in the form of examinations and independent work are made upon them. Then this coincidence creates an appearance of causal connection. In this matter only a careful search for the phenomena of dementia præcox, especially the proof of cataleptic and negativistic phenomena, as well as the appearance of characteristic peculiarities in the make-up of the hebephrenic can prevent the failure to recognize a psychosis.

The differentiation is less difficult when confronted with depressive states in the course of manic-depressive insanity. Nevertheless, here, too, one cannot use too much care. The excessive activities of the manic state may simulate real overwork. The previous history usually gives enough points of vantage to recognize this period of bustling activity for what it really is: not a stronger claim on the strength, conditioned by extraneous circumstances, but a sickly desire for activity, which spasmodically takes up now this, now that occupation with avidity, an inner restlessness, the raised emotional tone of which, often at first sight, discloses the pathological nature of this activity. The break down then, is not the reaction to the exaggerated demands on strength, but an alternation to the depressive phase. The indecision, the strong inhibition, the occasional self-reproaches, and the clearly defined feeling of illness, are other important points of recognition in diagnosis.

Prognosis and Course of the Disease.—The *course of the disease* is throughout favorable. Sufferers from acute nervous exhaustion recover, as a rule, with surprising celerity. But it is well to bear in mind from the beginning, the possibility of the *return* of similar conditions. If the return to those conditions that caused the overwork is unavoidable, the patient must be informed by the physician of the necessity of keeping safely within the line that separates healthy fatigue from unhealthy exhaustion. Change from physical to mental work does not suffice to prevent exhaustion, since both give rise to effects of fatigue though of different kinds. More important is a firm insistence upon certain periods for recuperation, which must not be too brief, nor set aside by *distractions*, and which naturally, are not to lead to exertion and exhaustion, avoidance of night work, and finally the regular pursuit of some exercise or sport, which must not be overdone. Only in this way can exhaustion of strength be avoided permanently.

Treatment.—Since the loss of strength is so important in etiology, we shall have to provide for the restoration of physical powers, especially when loss of blood, or serious diseases are considered the cause. But a systematic course of fattening will seldom be needed, since the patients usually recover quickly. In the beginning, recovery may be accelerated by preparations of iron, arsenic, and nourishing foods. In all cases, but especially in those in which mental overexertion caused the breakdown, it is essential that the patient should sleep as much as is required. Soporifics may be dispensed with in most cases. As soon as the patient is released from the pressure of work he cannot master, once he feels that he is no longer facing the daily increasing demands, sleep returns of itself. At most, in the beginning, one may attempt to relieve the patient of the troublesome feelings of physical restlessness, which keeps him from sleep, by slight hydrotherapeutic measures. I have seen good results from warm baths (93–96 degrees) lasting from half an hour to an hour or longer. The patients, whom I urge to read the newspaper, or some such literature, while in the bath, soon feel comfortable and are then able to sleep again.

If emotional stress of a chronic nature is concerned, the patient must at times be removed from his household relations. In simple mental overexertion, exercise in the form of sports is to be recommended, as forming a good counterweight to the continual strain upon the mind; travelling in medium or high altitudes often quickly restores the patient to health. But I should advise that a few days of as complete rest as possible should always be allowed to precede these journeys, that they should not begin at once with the most strenuous exertions, and that excess, of which there is so much danger in sports, should be avoided.

3. CONSTITUTIONAL NEURASTHENIA

Etiology.—General nervousness rests on a *congenital predisposition*. This circumstance will be quite obvious in the majority of cases, if one follows the life-history of the nervous individuals back to earliest *childhood*. Then we shall find, almost without an exception that at a very early age when external injuries are improbable, that there exist nervous symptoms the appearance of which is explicable only by assuming that the nervousness is a constitutional peculiarity of these children. It is far more difficult to obtain statistical proof of congenital degeneration from the previous history of the parents. Absolutely dependable figures will never be obtained. Only too frequently, the relatives deny the existence of nervous diseases, even where numerous illnesses have preceded.

By the mere ascertainment that nervous diseases have existed in a family, the problem of *psychopathic constitution* is not solved. For we see too often in gravely degenerate families single members developing normally, and on the other hand, very degenerate individuals come from apparently healthy families. In many of these cases, there would be no error in assuming that at the time of conception some injurious events had occurred. I call attention merely to the dangerous influence of intoxication at the time of conception. Even during pregnancy, diseases may so undermine the health of the mother, that the development of the embryo suffers.

The inborn predisposition, however, does not always lead to serious constitutional nervousness. As a rule we find, besides the hereditary tendency, *external causes*, which, added to the endogenous predisposition, first permit the phenomena of disease to appear. Where the seed of nervousness is already lodged in the child, care must be taken, even in childhood, to ward off all influences that might injure the nervous system. And especially in childhood, least attention is paid to this. Much that is supposed to be childish naughtiness is, in reality, nothing less than a sign of nervous predisposition, a storm signal, the recognition or neglect of which is of the utmost importance for the development of the unfortunate individual.

Among the most serious injuries I place a *perverted education.* Perhaps some sort of a talent, a not infrequent possession, has been forced unnaturally to an artificial bloom, or perchance some nervous conditions of anxiety or fear have been rebuked sternly instead of being treated carefully and quietly. Equally dangerous is excessive *yielding*, which early leads to pampering, attracts the attention of the child to its body and undermines the feeling that slight disorders may be overcome by the employment of some little energy.

With school a new mode of injury begins, the strong *demand on mental and physical powers.* The exertion demanded of the child by the school goes to the limits of what a healthy child can endure. Fortunately—at least from a medical standpoint—it escapes, by inattention, the harm that uninterrupted and concentrated attention, given for hours, would certainly entail. But for a nervous child, school so easily becomes a source of serious disturbances because, owing to the increased *liability to distraction*, a concomitant manifestation of nervousness, it becomes far more difficult, to follow the instruction with concentrated attention. Then comes the increased *tendency to fatigue* and finally, even disregarding the harm done by *imagined terrors* of all sorts, the often very distinctly manifested ambition. The school examinations and the exertion during preparation for the same, not infrequently furnish the final stimulus for the collapse. Even for grown people, moreover, *examinations or times*, during which unusual demands are made upon them, are the rocks on which the strength of the nervous founders most frequently.

Not only the school, but *life* itself, one may well say, life even more than school, expects too much of the growing child with the neurasthenic predisposition. Every walk through a large city, every walk in the open air claims attention, places before the unripe, inexperienced creature, new and difficult problems. If it could pass them by with indifference, it would protect itself against the danger. But the sensibility of the nervous person transforms every perception into a strong stimulus. The confusing wealth of impressions makes the child excited and sleepless. Happy the child whose danger is not increased by the folly of parents and teachers! Many of our books of fairy tales, the countless stories of robbers and Indians, which rebound from the sound thoughts of the healthy child, are poison for the nervous child, and the inclination of the parents, to satisfy the desire of their children "to see things" by visits to the theatre, and the horrible scenes of the kinematographs, may more frequently menace health, than the parents suspect. All these experiences, for this is what they are to the growing child, occupy his mind and thoughts ceaselessly, and overexert the brain, a brain not yet fully developed.

I believe, that the adult neurasthenic succumbs to actual overwork less frequently, than is generally assumed to be the case. For he breaks down, even before he has done too much. Certainly *emotional excitement* plays a large, possibly the chief rôle. A work, that may be mechanically performed, making claims only on the time, but not on the feelings of the nervous individual, is immensely better endured than a short piece of work, accompanied by intense emotions. Also in most other injuries, gleaned from the previous history of the nervous individual, we meet again and again, in a prominent place, the *emotions*. Conditions of exhaustion after physical diseases stand foremost among the *physical causes* of neurasthenia. Especially the infectious diseases and among these, just as in acute nervous exhaustion, typhoid and influenza the most frequently, bequeath conditions of general physical depression, in which various neurasthenic symptoms dominate the clinical picture. In women we find as causes of serious nervousness, too frequent parturitions and exhaustion from nursing.

Special consideration must be given to *sexual life*. Almost invariably the neurasthenic in his recital of what he believes led to the disease, will connect it with *masturbation*.

The difficulty of obtaining reliable statements concerning the frequency of masturbation will probably always prevent our getting figures that are entirely reliable, but my experience leads me to believe that only a very small percentage of men may boast of not having practised masturbation at some time in their lives.

Proceeding from this point of view, it will be seen that the significance of masturbation as a cause of nervous disorders should not be rated very highly, not even when it has been carried out to an unreasonable extent. For in such cases, as I assume in common with *Cramer*, it is already a manifestation of the psychopathic disposition or, occasionally, a symptom of a graver incipient psychic disorder, but not their cause. We find not infrequently that young people commit far greater excesses in normal sexual intercourse, which in spite of their nervous disposition are not followed by any injurious results.

The key to an understanding of this difference is furnished by observing that the nervous troubles begin only at that time, and only become particularly manifest, when patients have learned by reading pseudo-scientific works of the supposedly evil results caused by masturbation. The abominable habit of first portraying these consequences in the blackest colors, and of representing spinal consumption, softening of the brain, imbecility, impotency, loss of memory and of bodily strength as unavoidable consequences of masturbation, in order to afterwards recommend some expensive medicine or costly method of treatment as the only remedy, is daily claiming its victims among neurasthenics. And very often it is extremely difficult to shake the belief in these nightmares. But once the fear of later consequences has been removed, all troubles disappear, or at least, are considerably mitigated. This proves that not masturbation, but the fear of its consequences must be regarded as the cause of these phenomena.

Not always, however, are these manifestations due to books; frequently even a perusal of the advertisements in the columns of a newspaper, by their great number and exaggerated mode of expression will suffice to call forth in the neurasthenic a whole host of hypochondriac complaints. Encyclopedias also are often to be held responsible for similar troubles.

I have not met in all my many years of practice with a single case of serious nervous disorder, which could be unquestionably traced back to masturbation, nor, I may add right here, have I ever met one due to *continence*, either. It may indeed be rather hard for vigorous young people, particularly under the conditions in which they live today, to lead a life of sexual abstinence, but I am convinced that this abstinence is not productive of harmful results. In men, at least (and the question has here been discussed with regard to men, primarily) nature helps herself through spontaneous evacuation. *Nocturnal emissions* are indeed among the causes to which neurasthenics suppose their condition to be due. But there is no foundation for this belief, for too frequent emissions are either a proof of a particularly strong constitution, or, more usually a sign of a nervous disposition—a *symptom* and not a *cause* of the disease.

More recently those methods of preventing conception, connected with neo-Malthusianism, especially coitus interruptus, have come to be regarded as important causes of nervousness. It certainly is not a matter of indifference how copulation is consummated, and the strain on the attention in coitus interruptus may undoubtedly disagree with many and thereby give rise to nervous troubles. Whether this is true also of healthy individuals is questionable. In my opinion the question of sexual life has received more attention in the last few years than is to be desired in the interest of public health. The over-sensitiveness of nervous persons provides a fertile field for the development of all the dreadful portrayals. From their fears, which they base on their gleanings from books and which they therefore consider only too justified, develop the complaints and from these the conviction that everything that they read in books has come to pass, and that whatever is lacking, may be expected to occur very soon. The intense emotion, with which nervous persons react to every thing, is again and again the cause of the symptoms of the disease; and we should grossly misconceive the true causes, if we should take for granted all of those, which are indicated to us as such.

This is equally true also of countless cases, which may be traced back to chronic poisoning. Excessive use of alcohol, the poison most frequently used among us, gives rise, frequently, to nervous phenomena, besides more serious psychic disturbances. But experience teaches us, here too one must attribute to predisposition a degree of importance, that must not be underestimated. Not every man becomes a drunkard; here too a clearly defined predisposition is necessary, and this may be still more confidently affirmed of *morphomaniacs*. With very rare exceptions, morphine fiends are psychopathic and this is made manifest at the very first injection, which creates in them a quite unusual feeling of comfort. If, then, under the influence of protracted misuse, we see the nervous phenomena appear more and more strongly, we are not, as yet, justified in seeing in the morphine the cause of the nervousness. Coffee and tea also give rise to nervous troubles, but are not, however, to be regarded as causes of neurasthenia.

One of the most important causes of nervousness, trauma, is explained in a separate chapter.

Symptomatology.—*The "Psyche" of the neurasthenic.* We find neurasthenia in highly intelligent as well as in somewhat mentally deficient individuals; in both the tendency to quick fatigue, the same ineffectuality in regard to their hypochondriac disorders, the same inability, to fight off permanently by critical judgment, the uprising obsession-like morbid ideas, the same inclination to look upon the dark side of everything. The man with the better intellect will probably describe his condition better, analyse the individual symptoms better, be able to give a more sonorous and apparently more scientifically correct name to his troubles, but in its mode of origin and its symptoms, his disease picture differs in no wise from that of the mentally inferior individual.

The concurrence of nervous phenomena with *higher intellect* is, however, nothing strange or *accidental.* We find too often definite talents of all sorts among neurasthenics. Technical and mathematical problems are solved with great ease, the world of forms and colors, tones and speech is grasped and represented in artistic beauty; frequently we find the neurasthenics moving forward as the highly gifted pathfinders of civilisation. Usually this talent is shown very early, though the development of the child prodigy does not always fulfill the expectations of his short budding period. Many a seed dries up all too quickly, or is smothered by the mass of nervous troubles.

Nervousness and genius, are two phenomena of the same psychopathic predisposition. The same susceptibility and receptivity that makes the nervous life of the neurasthenic appear diseased, makes possible his unsuspected and bold combinations, makes his thinking and feeling deeper and more thorough, gives him the aspiring flight of the artist, the creative power of *genius.* Well may the stormy inner life, ever turned towards the highest problems, quickly exhaust the life power of the individual, and thereby cause the phenomena of a shattered nervous system to appear sooner and more easily. But the endogenous predisposition is common to both, the neurasthenic not specially gifted as well as to the man of artistic talents and creative genius. Therefore even where everything combines to protect the unusually gifted from the injuries of life, we see the concomitant nervous symptoms.

By far the most important manifestation in neurasthenia is the tendency to be *quickly exhausted.* The quiet, regular work of the healthy individual leads to *fatigue phenomena*, which are soon compensated for by sleep and rest. In the nervous individual, however, phenomena of exhaustion appear, even when the preceding work has been slight. Then it is more a matter of *subjective perceptions*, as seems to be proved by the fact that suddenly occurring important events may give the person who is apparently utterly exhausted, a quite unusual strength which makes him capable of great, and, if the seriousness of the external cause demands it, of prolonged efforts. But, at any rate, such phenomena are of rare occurrence. As a rule the neurasthenic goes to work with great eagerness, but soon *lags.* His energy resembles a straw fire, which quickly ends in smoke. *Beginning without executing* is characteristic of the neurasthenic's method of work. His *tendency towards enthusiasm*, however great it may be, soon dies down, and then he collapses all too easily.

His inclination to look upon, first and most prominently, the dark side of everything, makes him weak and undecided. As soon as a new task, entered into with great enthusiasm, begins to be difficult, the question arises, whether he can accomplish it at all, and immediately these doubts increase, until they rise, finally, as an insuperable mountain in the path of the nervous individual, and then it is characteristic that the time for rest, utilized by the healthy man to get new strength, often is the end for the neurasthenic. He cannot decide upon resuming work of which he sees only the difficulties, so he postpones the re-attempt to work. The more frequently this happens, the more undecided he becomes.

This is the moment, in which the neurasthenic, by *artificial* means, endeavors to prod on his energy, when he takes to *alcohol*, seeks to urge on his exhausted nerves by strong *tea* and *coffee*, to overcome nervous restlessness and discomfort by perpetual *smoking*, or to deaden them by *morphine*. And since similar conditions of failure appear again and again, the need for such agents repeats itself, until he can no longer dispense with them. The peculiarity is true of morphine far more than of alcohol, that most neurasthenics have the particular predisposition to experience the characteristic euphoria. Thus the longing increases for the drug, which for a short time at least, give him a feeling of comfort which is unusual to him.

The rapid failure, in spite of all his serious intentions, the swift escape of his enthusiastic inclination when face to face with harsh reality, is so impressed upon the sensitive spirit of the neurasthenic, that he soon trusts himself in nothing, *loses his selfconfidence* and finally meets every new task absolutely undecided and at a loss. The more keenly he feels his liability to exhaustion, the greater is the anxious discomfort, with which he approaches each new task, suspects difficulties, where there are none, and finally, in perpetually anxious expectation, breaks down.

The work of healthy individuals is affected, as soon as its regular progress is interrupted by *distracting* influences. When the strength of the influence is sufficiently great, the new experiences claim the attention so fully, that it is difficult to remain at the first task and escape the disturbing elements, but, as a rule, the healthy man knows how to force himself to return to the necessary work.

Not so the neurasthenic. Everything disturbs him, even if it can hold his attention but for a moment, external stimuli as well as his own thoughts. Here exaggerated sensibility plays a large part. A continuous slight *noise*, scarcely perceptible to a healthy person, becomes unbearable torture. A bright color distracts his attention. No one is so dependent upon external things as the nervous individual. If the paper is not smooth, if the ink is too thin, or too thick, the pen too soft or too hard, a window pane rattles, a faucet drips, any one of these suffices to make work impossible for the neurasthenic. Of course this dependence on external things, on the "devilishness of inanimate objects" ends at times in utter silliness. But just as certainly, it is often a phenomenon, whereby the patients suffer horribly.

The exaggerated sensibility shows itself most characteristically in regard to *daily events*. All of the petty and unavoidable annoyances, things that can not be prevented in a household, the least unpunctuality, the inexact performance of a task, a man appearing at the wrong time, a door closed with a bang, so distracts the patient that he can work no more. And this becomes all the worse, when not only everything that *does happen*, but also everything that *might happen*, has the same effect. One may safely assert that "what is coming" is more disturbing than the event itself. So, a nervous person may lie awake for hours, in anticipation that a train passing by towards morning, will awaken him, whose actual passing he may not even notice, if he is asleep. From the same cause, all that he has to do in the future, becomes the source of most painful nervous disturbances. If he must be host at or go to a party, hold a lecture, or pay a call, he thinks for days beforehand whether it will hurt him, if he will excite himself, if all will go well; and what is worse, the nights, too, are lost in brooding over this, so that finally, when it is actually time to do something, he collapses utterly.

Naturally, these peculiarities have their influence on his *mood*. The sensitiveness which transforms every pinprick of daily life into bitter torture, makes him irritable. If a neurasthenic flies into a passion at some harmless remark, or the banging of a door, this is due not so much to the effect of the stimulus, as to the fact, that he had even beforehand been in a condition of nervous tension, which exploded because of the event. The straw that breaks the camel's back must remind us that his back had been loaded to the breaking point before the addition of the last straw. If the experience seems too harmless, in the eyes of the healthy person, so that we find the great excitement of the nervous person inexplicable, we must not forget all that may have affected him formerly. This feeling of his *irritable weakness* makes the neurasthenic, who is usually only too well aware of his fatal inability to resist, very unhappy. And yet he can not command himself, because his sensibility is too great, the exciting events too frequent. So he wavers helplessly between tears and sulks, between enthusiasm here and despair there, in perpetual anxiety and restlessness, as to what will come, never enjoying life, unable to empty life's cup of bliss even once, conscious that at the bottom lie the bitter dregs that spoil his enjoyment.

The strong emotional tone of the brooding may become settled with extraordinary obstinacy in the thoughts of the patient, and lead to anxious, ever repeated fancies, which in spite of all resistance, appear again and again, and finally assume the nature of *obsessions*. Their presence may nearly always be detected in allusions, but because of the important rôle that they play in the life of many nervous individuals, a special description of them will be written.

The *physical manifestations* of neurasthenia are not very characteristic. The complaints are as manifold as the organs of the human body, and the trouble in comparison with the number of complaints, is objectively slight enough. The functions of the *sense organs* are scarcely ever seriously disturbed. Of course there may be *hypersensitiveness* towards *light* and *sound*, but this is a psychic, rather than a physical phenomenon.

In a like manner is the *failure of sight*, which occurs after long reading or looking intently, to be explained. First the patient sees very clearly, then the letters run into one another more and more, he feels as if he were looking through a veil, or through a slightly moving mist, finally he can recognize nothing more. Not infrequently the patients complain that muscæ volitantes dance before their eyes with unwonted persistence. The objective findings are always negative. The pupils are often very wide, which gives a peculiar swimming aspect to the look, so that a *neuropathic* eye has even been spoken of. The reaction of the pupils is active. Occasionally, a persistent variation in the size of the pupil is observed. The visual field is, on the whole, normal, but during examination, it sometimes decreases gradually. I attribute no great diagnostic value to this phenomenon, at least not as a criterion of fatigue.

The head is very often the seat of very strongly subjective sensations, among them *numbness*, which may increase to a feeling of the absolute impossibility of thinking, pressure which coming now from the outside is described by the patient "as if the head were fastened together with bands," now from the inside, "as if the brain would burst through the skull," of something gushing or dripping into the brain, of its bursting or being stabbed or burned.

In scarcely any neurasthenics are *cardiac disturbances* absent. Cases of *retardation* of the heart-beat are few, in contradistinction to many of *acceleration*, which may, at times, become so extreme that the pulse can scarcely be counted. It often appears associated with the feeling of pulsation in all the limbs; or as hammering in the ear before going to sleep, which is very annoying and may drive away sleep. The increased sensibility of the heart is manifested especially by the fact that every excitement shows itself by a very painful pulsation in the region of the heart, which betrays itself, as after physical exertions, by increased heart beats. Even irregularity is not very rare.

One of the most torturing manifestations is "*stopping of the heart.*" The patient suddenly feels a pain near his heart, and, at times, a feeling as if the heart were standing still, which takes his breath. It is usually a matter of a *heart beat not sufficiently strong to cause a pulse beat.*

The meaning of these phenomena has been the subject of hot discussion. On the one side are those who lay the greater stress on the nervous origin; on the other, many, especially *Krehl*, who warn us not to be too easily satisfied with a diagnosis of *heart neurosis*. Very often perceptible proofs of *organic* lesions manifest themselves later on in the course of the affection. This view is certainly correct, and it is therefore advisable, not to be too generous with the designation, *nervous palpitation*. But I should like to draw attention to the fact that the subjective disorders, in many cases, stand in an extraordinarily exaggerated relation to the objective phenomena. And if we consider, moreover, that not a few patients with serious heart trouble have but slight subjective disorders, the conclusion is evident that the nervousness is the essential thing after all. It shows itself more strongly in the heart, perhaps because an organic change lies at the bottom or is involved in the development.[1]

We meet similar difficulties in interpreting the extraordinarily frequent *disorders* of the *stomach* and *bowels.* If in a nervous person an examination of the gastric juice and of the function of the stomach proves the presence of too much or too little acid, this phenomenon may suffice to explain the disorder; but it may be—and according to my opinion as a rule is—only a symptom of nervousness; hyper- and hypo-acidity, then are but the consequence of nervous predisposition, which may affect the functions of the stomach in any way, as well as of the other organs. The correctness of this view seems to me to be best shown by the success of *treatment.* In many cases, all dietetic prescriptions, stomach pumpings, medicines, have but a slight and temporary effect. The patients move from sanatorium to sanatorium, from health resort to health resort. The range of foods they can stand becomes narrower and narrower. But if these same patients who react by violent pains in the stomach and by vomiting and diarrhœa to even the slightest deviation from their usual diet are not treated as dyspeptic but as nervous cases, the improvement in the gastric condition goes hand and hand with the general improvement of the nervous condition. I can certify, at any rate, that the number of patients in whom general nervousness is overlooked because of the obvious stomach and bowel troubles or is neglected as a secondary phenomenon is immensely larger than that of dyspeptics, who, only because of insufficient nourishment, show isolated nervous disorders or are nervous only because their digestive tract does not work properly.

Among the *subjective* phenomena the sensation of dullness predominates, an emptiness in the stomach forcing the patient to constantly take some nourishment. At times an insatiable *hunger* appears, which is quite independent of taking nourishment, or an absolute *lack of appetite* during which every attempt to take nourishment is followed by feelings of illness and vomiting. The slight decrease in the weight of the body is generally out of all proportion to the small amount of nourishment taken; but sometimes also a high degree of emaciation may appear.

Frequently there occur in severe paroxysms, which can not always be traced back to psychic excitement, *spasmodic pains in the region of the stomach*, associated with sharp heart-burn, belching and the vomiting of sour masses. The attacks are very painful, and because of fear of them the patient becomes more and more anxious in the choice of food. An examination of the stomach mostly proves that there is hyperacidity or occasionally gastric atonia. But the findings may be absolutely normal.

The patients are at one time persistently *constipated*, at another they suffer from *diarrhœa.* The strong influence of emotional excitement on the bowels is not wholly unknown even in the healthy individual. I mention only the diarrhœa often observed in war, sometimes during examinations. This phenomenon appears more strongly, of course, in the nervous person, in whom occasionally every project, going to a party or the theater, or a public appearance, causes attacks of diarrhœa. Occasionally in the watery excretions are found slimy substances and complete casts of the bowels (*colitis membranacea*). This form I consider also merely nervous disease, as is best shown by its dependence on the emotions, and its yielding to psychic therapy.

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[1] For peripheral vasomotor disturbances in neurasthenia, cf. the article by Curschmann, p. 934, ff.

An overwhelming desire to *urinate*, which may appear with every psychic perturbation and which, in case external circumstances prevent the patient from emptying his bladder, may be increased beyond endurance, has, as a rule, nothing to do with weakness of the bladder or of the sphincter. The *amount of urine* is not necessarily increased, but there appears frequently a considerable increase in the excretion of water, which may precede a lasting and scarcely satisfiable *feeling of thirst.* These cases of *diabetes insipidus* are not always easily distinguished from real *diabetes*, because on occasions even sugar may be temporarily excreted. But in these temporary *glycosurias* all other symptoms of diabetes are absent, so that actually this phenomenon, as a temporary symptom of neurasthenia is quite different from diabetes.

Among the earliest disturbances are those of oppression in breathing. Countless cases of *spasmus glottidis* in first childhood are to be definitely referred to an endogenous predisposition to nervousness. At a somewhat older age, but frequently also in childhood we have the development of *nervous asthma.* Its symptoms are so similar to those of bronchial asthma, that the distinction is frequently assumed to lie only in the greater dependence of the bronchial form on exciting influences and the more clearly marked catarrhal manifestations. This nervous asthma often disappears with surprising speed; the patient who has just been panting heavily and struggling for breath, can, an hour later, move about and go up stairs without much trouble. This quick recovery is, however, exceptional and, in spite of the fact that the cause is psychic, there is lacking neither cyanosis, nor the acute abnormal inflation of the lungs, nor *Charcot-Leyden's* crystals, nor the peculiar gasping râles throughout the lungs.

To the characteristic manifestations of neurasthenia belong *disturbances of sleep*, which we find even in early childhood in the form of *falling asleep late*, caused very often by ideas of anxiety or fear. The sleep is restless; the children talk in their sleep, turn from side to side, grit their teeth, pull at the bed covers. Not rarely is nocturnal *wetting of the bed* observed until puberty, but only exceptionally, in the pure nervous form, to the twentieth year and beyond.

One of the most characteristic forms of sleep disturbances in childhood is *pavor nocturnus.* Though I am convinced that many of the nocturnal attacks going under this name belong to epilepsy, it is very clearly also but a symptom of nervousness. The children spring up groaning, or screaming, with all the signs of a most intense terror; at times they answer questions without awakening at all. It is usually possible to dispel the fear of nervous children immediately, by turning on the light or calling to them. If the child says that bad dreams are the cause of his fear, this does not justify us, it seems to me, in accepting that statement as conclusive; probably the tone of the dreams is already a partial symptom of the feeling of oppression which finally leads to the outbreak of terror.

In the later course of life, the pavor nocturnus usually disappears, though this is not true of the other *sleep disturbances*, and the more neurasthenia evinces itself, the more the sleep disturbances step into the foreground. As a matter of fact they belong to the phenomena which we often can not reach, in spite of all our ways and means. The kind of sleep disturbance varies. Some patients have difficulty in falling asleep, roll for hours in bed, jump up, try by reading, eating, a bath, a poultice, to quiet themselves so that they finally fall asleep towards morning. Others fall asleep very quickly, sleep soundly for a few hours, during which nothing disturbs them, only to wake up frequently soon after midnight and remain awake the rest of the night. Others, yet, fall asleep, but for a short time, then lie awake for several hours, finally fall asleep again for a few minutes, say a quarter of an hour. Others sleep rather long, but always lightly, so that the slightest noise awakens them.

Very frequent is this peculiar phenomenon—suddenly, usually at the very moment of falling asleep, the patient feels a strong jerk, which may often, if not always, be recognized externally, by a short spasmodic twitching. This wakes the patient who now lies awake with a beating heart, in terror, and can not for a long time fall asleep again. Usually patients awakened in this way complain that they felt at the moment as if they were falling downstairs, plunging into an abyss, etc.

Dreams, on the whole, are a source of discomfort for the patient. They reflect the anxiety of the patient in a distorted and exaggerated picture. Nearly always their content is *terrifying*. Sudden death and murder, fire and earthquake, death and injuries, play the principal rôle in the dreams of nervous people. Very frequently the patient sees himself in school or at an examination, where, strange to say, the examination for University matriculation returns far more regularly than all the later ones; naturally all his knowledge has left him. Peculiarly painful is the feeling of being "*nailed to the spot*" which appears in nearly all the cases in which the dreamer believes himself to be in an unpleasant or a dangerous situation. He stands on the street half dressed, or untidily dressed, and sees a wagon, or a wild animal making for him. Or he hovers over an abyss, or descends stairs that suddenly fall in, under his feet. Or some one of his near relatives is in danger, while the patient is prevented by an overwhelming lassitude from helping. From these torturing dreams the patient wakes groaning, covered with sweat, with his heart thumping, and it generally takes some minutes, or longer, before he has quite overcome his fear.

The *contents* of the dreams often stand in very transparent relation to the events of the day; on the other hand I do not agree with *Freud*, who builds from them his material for proving the *sexual* genesis of neurasthenia. The re-interpretation, that the mental world of dreams must undergo at the hands of *Freud*, are so violent, so far-fetched, that in my opinion, they have no value as proof. I consider the fact significant that the dreams are fear-inspiring, but assign no importance, as a rule, to their content.

Beside the physical phenomena already mentioned all other disturbances fall into the background. *Sensory disturbances* of any clearly defined kind are absent, unless the entire aspect of the disease closely approaches hysteria, which as may be seen in the introductory remarks frequently occurs. But even where anæsthesias are absent, *subjective sensations*, paræsthesias of the limbs, pains in the back, pressure in the body and, as we have already said, especially headaches of every sort appear frequently.

The *reflexes* are throughout very active, but a *Babinski* toe reflex is never attained, though the strong twitching of the foot when the sole is touched, looks much like it.

For the sake of completeness we shall merely call attention to the *frequent trembling* of the hands, very evident at times, to the copious *secretion of sweat*, especially during excitement, and to all manifestations of poor *circulation* (cold hands and feet, goose flesh, hot head).

A phenomenon, to which we may describe closer relationship with the conditions of degeneration, than with neurasthenia itself, is "tic." Nervous starts, sudden shrugging of the shoulders, shaking, nodding of the head, movements of biting or chewing, sniffing, clearing the throat, coughing or lightning-like contractions of single groups of muscles, especially in the face, which like a flash transform the expression of the face into a grimace, for a few seconds, are very frequent concomitant phenomena of neurasthenia. They increase during excitement, and may entirely disappear in repose.

Finally *vertigo* must be mentioned. It usually appears in association with obsessions, for instance those of height or place, but may appear on the patient's entering hot rooms, looking at quickly moving wagons, and finally also disappear in repose. It is not increased, if the eyes are closed; many patients feel more secure as soon as they close their eyes—a proof that this vertigo belongs to the psychic rather than to the physical phenomena.

On the borderland between physical and psychical phenomena stand disturbances of the *sexual function*. Frequent *pollutions*, mostly, if not always, the consequence of the intense preoccupation of the patients with the observation of their own sexual life, an overwhelming desire to *masturbate*, *pros-*

tatorrhœa, that as a rule is usually mistaken for spermatorrhœa—which as a matter of fact does occur in rare cases—are the introductory troubles, soon to be followed by a host of others; pains in the *region of the loins*, where the patient supposes his center for the sexual functions to be, complaints about weak memory and lack of energy are further phenomena, which are made complete at last by *impotency*. If a patient attempts to cohabit there is either an ejaculatio præcox, or erection is entirely absent, or the patient is annoyed by strong erections, which give out at the very moment he attempts cohabitation, thus making it utterly impossible.

To mark off a separate form of neurasthenia, as *sexual neurasthenia*, has in so far less grounds in my opinion, since disturbances in potency are scarcely ever permanently lacking in neurasthenia. Experience shows that as a rule, as has been discussed in the introduction, the complaints are the result of misleading reading, and perverted instruction. Not infrequently such patients go to the physician with a whole pack of pamphlets of all sorts, with all the passages, applicable to them, underlined; notwithstanding all that may be explained to them, they call again and again on these witnesses. With the improvement of the general condition, the disturbances of the sexual organs occasionally disappear, but there are cases, in which they persist obstinately, because, in spite of the greater physical fitness, the fear of having injured one's self through masturbation, or of having become impotent because of continuous pollutions, remains afterwards as well, until this fear, also, is finally mastered, for the prognosis of these phenomena is not at all unfavorable, even though, because of the numerous and often most deeply inrooted prejudices, the physician can not always make an impression when brought face to face with the teachings of the unprofessional writers.

Prognosis and Course of the Disease.—The first traces, appearing in *childhood* are danger signals, announcing the later storms. Therefore the greatest energy must be used in influencing educationally and preventatively the nervously predisposed, even in his earliest years. What is missed in youth, can scarcely be made good in later years. The course further develops in greater or smaller fits and starts, but, as is to be expected from the nature of neurasthenia, as a constitutional disease, the patient will scarcely remain free from nervous disturbances. Or, to state it more clearly, he may feel himself free, but every excitement, every physical injury can, at a stroke, cause the entire mass of nervous troubles to reappear.

The *course of the disease* is strongly influenced by external experiences. But there are vacillations, in which it is impossible to make definite external grounds responsible for an apparent relapse, or to explain an apparent improvement by a favorable turn in the condition of his life, or the course of treatment adopted. Here there is clearly to be seen a close relationship with the periodic conditions of depression, which have not yet been sufficiently explained.

In spite of all his serious sufferings, which are frequently more unbearable than those with sound nerves are inclined to believe, there is, as a rule, no *danger to the life* of the neurasthenic. Yet we meet, not rarely, cases of suicide, for reasons already stated in the introduction. The neurasthenic commits *suicide*, not because of the suffering he undergoes, but for fear of those that will come. *The fear of softening of the brain*, of becoming insane, of *cancer* and consumption has put, without any more material reason, a revolver into the hands of many a man. Also the suicide can occasionally be explained as the result of the feeling that the victim is not equal to a change in his life, even a favorable one, a great work, or an examination.

Without doubt to this category belong the not infrequent cases, in which *adolescents*, from fear of punishment, or from excitement over a reproof put an end to their lives. The sensitive neurasthenic is more affected by events than the healthy man, and has not the vitality or the equanimity of the robust; thus he breaks down, and believes that death is the only way out of his troubles.

Differential diagnosis.—The diagnosis of *constitutional neurasthenia* is, on the whole, not difficult. The patient generally begins his complaints with the fact that he has always been nervous and suffering. But this statement does not absolve us from the responsibility of making a thorough physical examination. Even in the cases in which the patients assert that they have always been well heretofore, one can not be too careful. A diagnosis of the disease as "nervous" is justified only when every physical disease has been excluded.

The exhaustion, which appears as a concomitant of various *physical diseases*, such as carcinoma, diabetes, anæmic and leukæmic conditions, reminds us closely of the exhaustion of the neurasthenic. In *diabetes mellitus*, it must be specially remarked that considerably increased urination without sugar, is not rarely a nervous symptom, and that, on the other hand, occasional alimentary glycosurias are observed in neurasthenics, without justifying one in speaking of a real diabetes.

The most frequent error made in diagnosis is the overlooking of incipient mental disturbances. Among these, *cerebral softening* stands first. The appearance of nervous exhaustion in middle age in a person who has until then always been healthy, must awaken a suspicion of a serious disorder, and then a careful examination will soon disclose one symptom or another of brain disease. A very important help in diagnosis is *cytological* examination, which may be used in deciding important cases, with great success.

In *adolescents*, we find difficulties in distinguishing neurasthenia from *dementia præcox.* This develops very frequently as a hypochondriacal moodiness. At the beginning of the disease, the troubles are told, with considerable emotional stress. The patients complain about difficulty in thinking, a deadening of the intelligence, anxiety and restlessness, irritability, and fatigue, and, as a rule, combine these complaints, with those of physical troubles.

Further development usually leads to fairly quick decision. But one will not always be able to wait for months. I have been very frequently enabled by clinical observation at a hospital to come to a decision as to patients whose affection could not be correctly diagnosed at the time they were examined at their home or in the office since within the hospital the stereotyped positions and movements, as well as the extraordinary peculiarities of the hebephrenics were quickly revealed.

More frequent is the confusion with *periodic depressive states.* Since both affections develop on the ground of constitutional degeneration, we are not assisted by the ascertainment that the patients were even formerly somewhat nervous and sensitive, easily fatigued and inclined to hypochondriacal complaints. Even in the external symptoms, the more or less clearly defined inhibition, the strong feeling of inefficiency, the two diseases resemble each other; and if there are added, as manic components, occasional irritability and excitability, the clinical picture of neurasthenia resembles so closely the picture of periodic depression, that it is possible to distinguish them only by the subsequent course of the disease.

In advanced age, there are scarcely any special difficulties in distinguishing neurasthenia. At most, the symptom complex of arteriosclerosis, may simulate the clinical picture of neurasthenia. But here, previous history, the increased blood-pressure, and the perceptible symptoms of hardening of the arteries, are important and generally sufficient distinctive points.

Treatment.—In the treatment of nervous conditions, we shall have to proceed quite differently, according as we wish to treat the *symptoms* or the fundamental *disease.* The patient wishes to be rid of the manifestations that trouble him, but the physician must go beyond the manifestations and consider the improvement of the disease as a whole. If, for instance, I systematically wash out the stomach of a *neurasthenic*, who, every time after eating, complains of pains in the region of the stomach, together with hyperacidity, I can get rid of these difficulties, but the condition of the disease really remains the same—is even aggravated—inasmuch as the patient, because of this special treatment, thinks his stomach trouble particularly serious, and makes it the foundation for new hypochondriacal fancies.

Still less does **local therapy** prove effective in *hysteria*—and naturally enough. But this does not mean that it is not often necessary that the most troublesome symptom must first be got rid of. An hysteric aphonia, a contracture, a painful point, hinder the patient in every activity, wherefore he must be relieved of these symptoms before general treatment can be successful. But in every case the purpose of the treatment must remain in the foreground, namely to rid the patient of his disease, so perfectly that the appearance of new symptoms, is, as far as possible, prevented, and that, when they do appear, the patient himself may know how to act with regard to them.

In the conception which I present, that neurasthenia as well as hysteria is a *constitutional state*, the word cure will have to be used with great caution. The sensitiveness of the nervous system will always remain, but it is in very many cases possible, to bring the patient into a condition that practically resembles *recovery*. Since it is a matter of psychic disease, *psychotherapy* has precedence over all other remedial agents. And in all cases—this must be clearly comprehended—in which operations, mud- and sunbaths, medicines, or miraculous elixirs have been followed by surprisingly great successes the apparent cure consists only in the patient's firm confidence in the effectiveness of the remedy used.

Surgical treatment, is, in the majority of cases, a mistake. It is clear enough that where any local disease, for instance, a persistently painful joint, affected with a "joint-mouse," would necessitate an operation even in an individual who is not nervous, the concretion must be removed. But in every case, the question whether the operation is really necessary should be asked more seriously than usual. The time has fortunately passed in which every nervous woman had to have her womb curetted or pessaries inserted, or had to undergo ventral fixation, or even castration. Nevertheless, we still find too many patients, who have undergone serious abdominal operations, have had the appendix removed, or have been operated upon for ulcers of the stomach or gall-bladder troubles, though no serious disease was ever present.

Occasionally, this is due to *mistakes* in *diagnosis*, mistakes that are really difficult to avoid, but more frequently to a confusion in cause and effect. This is true also of non-operative procedures (pumping out the stomach, long-continued electric treatment, catheterising, etc.). Though sometimes a good result is reported, I am far from considering all these procedures harmless. The danger lies in the fact that the attention of the patient is attracted to his troubles instead of being distracted from them. And then we perceive, from the relapse that follows the first improvement, or from the spreading of the troubles to other organs, that the actual foundation for the complaints is neglected for their *apparent* cause.

One need surely waste no words on the wisdom of never resorting to *fictitious operations*. Beside their ineffectuality—for every symptom got rid of, one or several new ones appear—it is extremely dangerous for the physician, if the patient sees through the comedy, which, moreover, is an unworthy game to play with him, and thereby loses all confidence in the physician, which in this case would be entirely justifiable.

Confidence in the physician is the most essential condition in the treatment of nervous people. In order to win confidence, it is not sufficient to content one's self with a superficial examination, even when obvious hysterical manifestations are present. The examination must be made rather with the greatest care, must include the entire body, since we must always bear in mind the fact that very frequently psychogenous symptoms appear in organic diseases. It is in this very respect that most caution must be used.

A careful examination of the patient's condition must, in every case, precede plans for the cure, not only because of the necessity for avoiding a false diagnosis, however important this point may be, but also to win his confidence. The nervous person is more mistrustful, more inclined to hypochondriac self-observation than any other patient. If he is to believe the physician's assurance, that the heart, stomach, brain, are sound and well, he must see and feel that because of the thoroughness of the examination, nothing can escape the doctor's observation. Only then will he permit himself to be really pacified. To the examination of the patient's condition belongs further, a *knowledge of his personality*, which must not be satisfied with a superficial ascertainment of the most important events of his life. The physician must know what sort of mind the nervous individual has, since he must inject the personality of the patient as the most important factor into his therapeutic measures. Only on this foundation can he make any progress against the *disease* itself.

Every treatment of a nervous individual must be at once *general* and *specialized*. For general treatment, the chief dependence must be upon *removal from his home surroundings*. It is not saying too much to assert that half of the treatment is accomplished by separation from his family. The reason lies partly in the separation from the petty annoyances of everyday life, which are mostly far more disturbing to him than serious catastrophes—partly in his greater susceptibility to the influence of the physician. Whether this separation is to be combined with an attempt to *fatten the patient*, depends less than was formerly thought on his physical condition. Of course it will be necessary to strengthen the anæmic individual, or one who has had insufficient nourishment, but it would be a mistake to stake all one's hope on simply physical strengthening and increase in weight. For the strengthening of physical forces, arsenic has proved to be very effective. It may be given in the form of Fowler's solution, in pills, in ferrous arseniate or Levike water. Favorable results—probably not from psychic reasons alone—are produced by *general faradization* of the entire body, and especially, by massage. I can only advise urgently, that the massage be combined regularly with active and passive movements of all the limbs, or with systematic physical culture exercises, since with the feeling of greater *physical ability*, that of psychic ability seems to grow.

Of *hydro-therapeutic measures*, baths and wet packs (Einpackungen und Abklatschungen) are mainly to be considered. The addition of carbonic acid and pine needle extract—Fluinol is cleaner and therefore more pleasant—

generally adds to the effect of the baths. Whether this is due to other than psychic effects is doubtful.

For *sleeplessness* I can only recommend that the baths at a temperature of 94–96° F. be of rather long duration, one or two hours. Of the supposedly weakening influence of long baths I have never seen any evidence. In order not to be worried by brooding, during the time spent in the bath, the patients may quietly read a newspaper or harmless books. Then the soporific effect is often more powerful than that of many chemical soporifics. Wet packs, too, which should not purposely be extended at first beyond half an hour, but later may without hesitation last an hour, or more, have a very favorable influence upon sleep. But with all these remedies, one must remember primarily, that they have not the same effects in every case, and that occasionally, the nervous restlessness may even be increased. But least of all can I accustom myself to the other countless procedures, in so far at least as cold water is used. Most nervous people are injuriously affected by the momentary shock unavoidably entailed by the use of *cold* water. In all cases, in which hardening of the body is concerned, it is recommended to begin with quite lukewarm water, and gradually advance to cold.

To the treatment of nervous people belongs *supervision of their activity.* The best thing is to prescribe for them, at least at the beginning, an exact division of the day, which besides other benefits, has the advantage of accustoming the patient to a *dutiful execution* of all orders. No instruction as to how this division may best be arranged, can be given, since the directions must be arranged according to the personality of the patient and the physician. The greater the confidence in the physician, the more quickly he will be able to proceed.

No rule can be given as to how the local symptoms are to be treated. As a rule it would be advantageous to employ as *little* local treatment as possible. But this does not mean that local symptoms are to be neglected, or that it is sufficient to designate them as *imaginary.* The patient will be thankful if one agrees with him that he really has neuralgic pains, can not move his arm, and can not overcome the stiffness of his leg. If one yields in this, he will not be offended if one explains to him that there is no organic change as a foundation for these phenomena, and that the less attention is paid them by patient and physician, the more quickly they disappear. This is true especially of such nervous phenomena as are not continuously present, e. g., nervous asthma, digestive disturbances, exhaustion, headaches, etc.

In localized paralyses, local treatment may, at times, result favorably, especially with patients who have not as yet experienced the magic of the apparatus; for instance, the static machine. On the whole, however, too much value must not be placed upon all these manifestations, and it must be remembered that getting rid of a symptom is not the main affair. Only when the patient knows that his psychic symptoms may arise without an anatomic foundation, he will teach himself to avoid the constant danger of the return of similar, or the appearance of new manifestations.

Of all the phenomena, none so frequently demands particular attention as *sleeplessness.* The persistency of this symptom places upon us the duty of being very cautious in handing out *medicines* because of the danger of the patient's acquiring a drug *habit.* However desirous one may be of casting aside every chemical soporific, their use can not be avoided in cases of especially troublesome sleep disturbances, in order to induce sleep, but first all other measures, and particularly the hydro-therapeutic, should be tried as well as simple changes in the mode of life; sometimes good results are obtained from early dining, sometimes from eating directly before going to bed, or even during the night; at others, physical weariness or complete rest should precede the time of going to bed.

Only when all these methods fail should soporifics be tried. I prefer in such cases paraldehyde and hydroxide of amylene in not too small doses, 6–8 gr. of the former, 4–5 gr. of the latter, to the more specific soporifics, not only because of the manner of their working—they are meant to put the patient to sleep, not to drug him and keep him asleep for any length of time—but because patients, as a rule, do not become enthusiastic about these remedies. To give morphine is quite futile, even not considering the danger of the drug habit; it generally keeps the patient awake, though he may find this waking stage very pleasant. Of soporifics, in cases of serious sleeplessness, I prefer trional to the favorite veronal. It works more surely and enduringly, though one can mostly do without it, but one thing must be especially noticed: occasionally a powder of bicarbonate of sodium, or (this is recommended specially for hospital practice) a solution of methylene blue or eosin, is successful where other drugs have failed. I remember an hysteric woman, in whom even the strongest soporifics in large doses procured sleep no longer than possibly two hours, whereas she could sleep the whole night through very well indeed, on one- to two-tenths of a hypodermic syringe, filled with nihiline (common distilled water).

Occasionally, *sedatives* are indispensable in coping with *excitability.* The patient has a feeling of greater security, when he has taken some medicine, and this feeling has a restful effect. The tests of pharmacologists, it is true, rob most of the drugs recommended as "strengthening the nerves" and as sedatives of their halo and prove that the effect is wholly, or, for the most part, merely suggestive. Yet in the *valerian preparations* there seems to be, besides the suggestive, an actually quietive effect. This is true mainly of valerian tea, which I prefer to the other preparations as valerian tincture, as well as to validol, valyl and bornyval. Not infrequently the patient is quite satisfied by the feeling of having a soothing potion in his pocket, which he may take in any emergency, to put a damper upon his excitability and feel himself secure.

The favorite drug is doubtless bromine and with full justification, because bromine actually lowers irritability, as the psychological experiments of *Löwald* proved. Only one must not believe that a great deal can be accomplished by small doses; 2–3 gr. bromide of potassium or bromide of sodium—*Erlenmeyer's* mixture with bromide of ammonium is scarcely more effective—can unquestionably be given for a considerable time. But however indispensable bromine often is, the same holds true of this, as of all other methods of treatment: they are suited to combat a symptom only, not the disease.

Hypnotism needs special mention. It is remarkable how difficult it is to overcome the *prejudice* against this method of treatment. In the hands of the skilled physician, it is a very valuable means of treatment, and though dispensable in many cases it is not so in all. I have seen excellent results especially in desperate cases (that had been treated for years) of compulsory thought, anxious states of all sorts, also in persistent headache, sleeplessness, etc. I say quite frankly to the patient that it is merely a matter of convincing himself. But to teach them that their troubles, objectively considered, are harmless, I try to awaken in them belief in the possibility of recovery, and try to point out the way by which such phenomena may be avoided in the future. The greater concentration of attention during the slight degree of hypnosis, that I generally employ, and that, in my experience, suffices, makes it easy to teach the patient and to help him. Whether we use as suggestive means, the faradic current, a drug or hypnotism, is all the same in the end. I merely find that hypnotism is more honest, and, in most cases, more effective.

Formerly I opposed the use of hypnotism with hysterics. My additional experience has induced me to change my opinion; only in the use of hypnotism, as well as of all other methods of treatment, one must remember, that only the symptom, not the disease, is gotten rid of. The fairy-tale that hypnotism still further paralyzes the energy of the patient can be seriously believed only by those who have never tried it. I can assure them, that many people, on whom all other methods of treatment were tried in vain, finally regained their life energy by means of hypnotism.

Of all forms of treatment, I am least in sympathy with the so-called *startling* (Ueberrumpelung) and *brusque* methods. If one lifts up a patient who thinks she is unable to stand, and then drops her upon her legs, she may occasionally be suddenly able to stand. Then the efficiency of the physician appears in a favorable light. But nevertheless the method is wrong. If it fails, which happens frequently, far more frequently than it succeeds, the physician has exposed himself under the very eyes of the patient, and lost the confidence that is essential to his treatment of her. If it succeeds, the physician has merely got rid of a symptom, and not helped the patient in his disease.

But I think the much used method of torturing patients by means of painful agents—the *faradic brush* is a special favorite—still worse. In this method, there is still a trace of the old prejudice, that the patient is *simulating*, and does not wish to recover. Occasional transient successes, which have been attained by this method also—what method would not occasionally work wonders with hysterics—prove nothing as to its value. Whoever cannot rid himself of *prejudices* in regard to the nervous, who has not the *patience* to listen to their complaints, in so far as it is necessary, and who does not from the first make it his aim to *educate systematically* the patient towards health and to combat their autosuggestions, had better forego the attempt to treat nervous cases. And here, I shall permit myself a piece of advice. Not infrequently the physician takes great pains with a patient all in vain, because the patient can not command confidence in the physician, a confidence that is the indispensable supposition in the treatment of nervous patients. If the physician notices this, he should give up further treatment, and not think that his authority has suffered. There are often imponderabilia, which give to one man the success denied to another.

The *plan of campaign*, that must be made at the beginning of the treatment of a nervous individual, can not be too comprehensive. It must not be confined to merely getting rid of the momentary condition, but must reach as a preventative far beyond it and be directed against the reappearance of the old, or the appearance of new phenomena of the disease. Correspondingly simple is the procedure in the case of acute nervous exhaustion, far simpler than in all other forms of nervous diseases.

In *neurasthenia*, the patient must first be quieted and confidence in his physical and mental functional capacity must be restored to him. For this, systematic *training* is necessary, and though some sanatoria do good by accustoming the patient to enjoy the rest necessary for him and train him to sit still and to rest, still this must not be the ultimate aim. The patient, on the contrary, must gradually begin work; physical exercises, and various sports, finally bring him as near as possible to health.

With *hysterics*, in the majority of cases, occupation for the physical and mental powers is desirable *a priori*. As *little treatment* as possible, since the patient considers it merely in the light of confirmation of his unhealthy ideas. The tendency of hysterics to become enthusiastic, very frequently makes it possible to furnish them an occupation, in which they can advantageously utilize their excessive emotions. They are possibly not always the persons best adapted to social activities, but under proper guidance, they can do much good and then often find the satisfaction they need to distract them from unceasing self-observation.

Finally a *warning*. That marriage (I am not considering the possible disadvantage to posterity) brings improvement to many nervous persons is unquestionable, but this improvement lies in the greater regularity of life, the possibility of expression, distraction from personal cares by the little cares of the household, though these, at times, make their condition worse. To advise marriage, in the hope that sexual satisfaction will have anti-hysteric results, may be regarded as a direct proof of a complete lack of an understanding of nervous conditions.

4. THE TRAUMATIC NEUROSIS

The development of the disease picture that we now call *traumatic neurosis*, goes back to the year 1866, in which *Erichsen* first turned attention to phenomena that develop in connection with injuries in the realm of the nervous system. *Erichson*, like other authors, increased the knowledge of the details of the disease, but only in 1881, special emphasis was laid by *Moeli* on the psychic phenomena of traumatic neurosis. The further progress of the conception is shown best by the view represented a few years later by *Charcot*, that the entire disease picture was that of hysteria. This was opposed by *Oppenheim*, who invented the name, *traumatic neurosis* and assigned to it a special character. The final point in the entire development is represented by *Kraepelin's* nomenclature, who by the designation of *terror neurosis* (Schreckneurose) transferred the cause of the disease wholly to the psychic realm and made it independent of any injury.

Today it is regarded as a generally known fact, that there is no special form of disease—traumatic neurosis—rather that under this designation both neurasthenic and hysteric clinical pictures are understood to be in very close association and admixture. Even so, it is helpful to describe them apart from all other psychasthenic states, because a peculiar color is given it by its connection with our *industrial accident legislation.*

Under the name traumatic neurosis are to be understood the *psychic disturbances of non-organic nature that develop in connection with a trauma.*

Etiology.—The definition is based on the fact that the disease is associated with a *trauma.* The *seriousness* of the injury that has occurred is far less significant than was formerly thought, though a certain relation of dependence is only too natural, because of the dependence of nervous phenomena on psychic impressions. I place great value, however, upon a definite warning not to cling too closely to this natural and obvious relation between the seriousness of the injury and the seriousness of the manifestations of the disease, for, in the first place, we are not in a position to set up an even approximate measure of the seriousness of the injury. A slight stiffening of the fingers in a pianist or a clock maker is a more serious injury than the loss of several fingers to a farm hand; the psychical shock is also more serious. But besides this, experience has shown us, that frequently serious organic diseases may be attached to injuries that appear slight externally. In this matter, special difficulty is caused by injuries of the skull. Since I have seen in several cases, which have been confirmed by operation, that with scarcely perceptible laceration of the skin, serious fractures of the vault of the cranium and extensive hemorrhages in the brain, and that even severe lesions of the brain substance may appear without important wounds in the soft parts and apparent fracture of the skull, I have become very skeptical, when the evidence of the witnesses testifies to the injury as slight. I distinguish—though this needs no special emphasis—the *organic* consequences most sharply from the *nervous* ones; but the experience mentioned would alone suffice to protect us from excessive valuation of the objective proof of the extent or seriousness of an injury, in our view of the consequences of an accident.

More important than the *objective* injury is the *subjective.* I saw in an engineer, who succeeded in stopping a train before collision with another, in a man who was only saved from a red hot furnace by the interference of courageous fellow-workmen, and in a young girl, whose clothes had caught fire, though without the slightest external injury, the identical symptom complex of traumatic neurosis develop. Therefore I believe that *psychic excitement* at the moment of the accident is the most important factor to take into consideration. Likewise, I do not consider it right to designate these states as *terror neurosis*, because terror is only one of the psychic causes, and everywhere steps into the background, where chronic emotional states are concerned; moreover, because by the designation, terror neurosis, the connection with the *accident*, and therefore with our *laws relating to accidents*, is obliterated.

I have just remarked that the affection is not always associated with an acute nervous shock, and thereby suggested that it may often develop quite slowly. Very frequently in the beginning the patient complains but little. Then gradually *anxiety as to the future* begins. The patient who sees himself no longer equal to the exertion demanded by his tasks, becomes tired sooner and easily feels pains; then he begins to ask himself, what will become of him, if the disease progresses. On the thus prepared basis of anxiety as to the future, the *accident insurance laws* now begin to show their unhealthy effect. They should be so drawn and executed as to pacify the patient, who should be able to say to himself, that under all circumstances, he is to be saved from dire need. But this beneficial effect is often lacking, partly because the patient's accident insurance is too small, especially when he has a family, but still more frequently, because he has to make a tiresome fight to establish his claim to the insurance.

Strümpell believed that he could assign special importance to the "desire to make money out of the accident." Of course, it seems to many people very enticing, to be able to draw their money every month, though it be but little, rather than to earn an actually larger sum by hard work. I believe, however, that the desire for compensation is not nearly so important for the development of the disease, as *Strümpell* and others who agree with him assume. We find the very same symptom complex even in people who care nothing for this attainable recompense for their injuries. I saw, for instance, the phenomena of a typical traumatic neurosis appear in a very wealthy man, who was to receive a trifling income from a private insurance company, when an intimation of simulation and *exaggeration* was expressed by the insurance company. Anger about this, had the same effect as terror in some men, and covetousness in others. The phenomena disappeared when the patient, upon my advice, sacrificed himself and gave up all claims to the damages. Similar cases, if not always so clearly marked, have been observed by others, and so it is perfectly correct to look on the desire for compensation as only one, not as *the* cause of the affection. The refusing—in many cases one may justly say tricky—attitude of the insurance companies, whether they belong to the state or are private concerns, gives rise to a peculiar mood in the injured man. He becomes indignant at the *mistrust*, worries as to whether he will receive sufficient compensation, whether he will be correctly judged by the physicians. On this ground, there develops the *fight for the money*, with all its psychic concomitants and consequences.

At the moment of the injury, one can not foresee, what psychic phenomena will follow it. If the injury has physical consequences, especially such as to *disfigure* the patient or cause him permanent *pain*, this also is significant for the development of the disease. Now it is physical consequences, now the terror experienced at the time of the accident, now the anxiety as to the future, now the fight for the insurance, by which the first impulse to traumatic neurosis is given. The variety and overlapping of the causes prevents us from stating a rule, according to which the phenomena may be established in certain relations to the causes; still more, the fact that terror from an accident is quickly overcome by one man, and not by another; that one man soon accustoms himself to physical consequences, while another can not. So observation forces us to the conclusion, that the *predisposition* of the sufferer is of remarkable importance. *A nervous predisposition* must be regarded as the foundation for the development of every traumatic neurosis. The more serious the psychopathic predisposition, the more quickly and distinctly the nervous consequences of accidents appear, the more obstinately the emotions take root, and allow the disease to reach its richest bloom.

There is still another cause to mention, the importance of which can not be insisted upon too earnestly—*the influence of the physician.* In an extraordinarily large number of cases, nervous phenomena develop after accidents, just as the usual neurasthenic phenomena in non-traumatics, in connection with a stupid remark by the physician. The words "concussion of the brain, internal injury, serious case" become the starting-point for the hypochondriacal complaints of the patient, and all later attempts to quiet him will fail to do away with the once established influence of this terror phantom. Precisely in the case of accidents, the physician should, for *prophylactic* reasons, from the beginning of the disease, treat the case, when face to face with the patient, as slightly as possible, and not be too ready to attest to his *inability to work.* The excessive liberality in ordering patients to bathing-places and health resorts is dangerous. Of course I do not mean that necessary procedures and arrangements should be left unmade. But in addition to the possibly desirable recuperation appears the danger of a too deeply rooted idea of the seriousness of the disease and the additional danger, that the patient may take pleasure in idleness, and at health resorts accustom himself to a luxury not corresponding to his usual circumstances.

On the other side—and I may say frequently rather than seldom—the aggravation of traumatic neurosis is to be traced back to *examinations by that sort of physician,* who, from the first, meets every sufferer from an accident with the greatest *suspicion.* That the patient who does not correctly estimate his disease, and thinks himself injured in regard to his just interests, becomes excited and feels himself offended by the intent to deceive imputed to him, is a thing of frequent occurrence. And then, if he portrays his complaints more and more vigorously, and resorts to exaggeration and simulation, this is a defensive measure against the wrong done him, and is, if not defensible and commendable, at least easily intelligible. Occasionally the patients confess, that they exaggerated because they had expected or felt, that they were not being absolutely believed, and had hoped that after all deductions had been made, at least some faith would be placed in their troubles, as they actually had serious cause for complaints.

Symptomatology.—The close relationship of traumatic neurosis with neurasthenia and hysteria, gives us reason to expect *à priori* that all those manifestations that we have observed in psychasthenic states, will be found here also. Naturally, every attempt to discover *objective* symptoms, because the frequency of exaggeration and the importance of the decision upon the claims for compensation make judging the case a serious task, is frustrated by the fact that a *psychic* affection is concerned.

The more conscious one is of this, the less intelligible it becomes, that so little attention is paid to the *psychic concomitant phenomena.* In the contest between those who suspect simulation everywhere, and those who consider it a rare thing, the hard word has dropped, that the number of simulators stands in inverse proportion to the psychiatric knowledge of the man who judges. Nowadays, the conviction of the importance of psychic genesis, if it has not been wholly driven home, has at least progressed so far, that one may designate as a mistake a decision about an accident neurosis without exact investigation of the psychic status.

The most important phenomena, for us, lie in the psychic realm. Thereby the patients are distinguished from neurasthenics and hysterics, that in their ideas, the accident and its consequences play an exaggerated part. To the consequences belong, also, anxiety as to the future, excitement about the fight for insurance, and the desire for compensation. Whereas the neurasthenic, in spite of all his morbid scepticism, tries every new means, every method, even permits himself to be the subject of the most venturesome healing processes, entrusts himself to every quack, the victims of accident neurosis, in many ways, oppose all therapeutic measures on the grounds that they are futile. And even where they submit to treatment, they do it with inner resistance and the prejudice, that only a reduction of their annuity and not a restoration of their former functional capacity is to be attained.

Their *mood* is absolutely irritated or depressed, full of cares and anxieties. The patient becomes more lively, only when the conversation turns on his accident and his fight for the insurance. The older the case is, the more frequently medical investigations have preceded, the more lively and larded with technical expressions, is his presentation, which he mostly supports by producing his "documents." The physicians, in his opinion, are divided into two classes—as in the case of the querulous man—into those favorably inclined to him, and those in the service of the accident insurance company. It is virtually impossible to force the patient to an objective conception of the case. Every contradiction irritates him, a careful examination gives rise in him to the suspicion that one wishes to weaken his claim, and makes him rebellious against the physician. Thereby such patients in hospitals often get to be most annoying, because they are always finding fault with something, scolding and ranting, and thus infect the other patients.

All thoughts and feelings, especially in cases of long standing, turn only about the accident and its consequences. The patient returns to his accident again and again; he revels with peculiar contentment in his troubles and the portrayal of his difficulties that arise in his fight for insurance. Care for the family is strongly emphasized in the complaints, but, as a rule, the patient thinks of his family none too often, as his own "fate" stands too pronouncedly in the foreground of his thoughts.

Often this one-sidedness, which together with the portrayed inclination to instigate mischief and calumniate, constitutes virtually a change in character, is combined with a clearly defined *depression*. The patients sit sadly around the house, take no interest in anything, speak little. If anger or hypersensitiveness to the little pinpricks of life causes an explosive burst of rage, the patient breaks down immediately afterwards, and laments his tragic fate in whining tones, and is often bathed in tears. Only rarely one meets with such a resigned and courageous sacrifice of the enjoyments and happiness of life, as we often find in cases of hysterical diseases.

The *functional* capacity is usually *diminished* considerably. Every exertion calls out feelings of fatigue, of long duration. Even in the mental sphere, and perhaps most clearly in this sphere, one sees strong interference with the working ability. The patients become *inactive*, find great difficulty in performing new tasks, even though these tasks may make no demands worth mentioning, are distracted, and easily turned aside; even after a short time, the work progresses with difficulty, and the patient is soon entirely exhausted.

The frequent complaint about *disturbance of the memory*, is, for the most part, merely a *disturbance in the power to receive and retain new ideas.* The old knowledge, as a rule, remains unchanged, though it may not be so readily at the disposal of the patient. On the other hand, it is often very difficult for him to take in new impressions. The typical "accident" patient, as soon as he receives a command, or is summoned for examination for another day, immediately draws out his note-book, in order to help his failing memory by writing it down. The test of this power to receive and retain new impressions is best made by naming a couple of words or a number, which the patient must repeat after one or two minutes, during which his attention is turned aside by other questions. Very infrequently is this task performed easily, if a serious case of accident neurosis is before us.

The feeling of functional inability in the mental realms is the most annoying of all phenomena, especially for a rather intelligent person, since he can only pull himself together with difficulty for an energetic attempt to work. The more frequently he fails in such an attempt, the stronger becomes his fear, that he will soon no longer be able to do anything at all, until at last, the patient has completely convinced himself that he is absolutely and finally played out.

A phenomenon, by no means rare, is the increased *capacity for fear*, which is wont to attach itself to the original cause of the condition. Thus the man injured by an electric car, is startled, whenever he hears a gong, the man injured on a railroad, whenever he hears a locomotive whistle. This anxious state recurs in his *dreams*, in which the terrible events, distorted and exaggerated, may return with painful obstinacy. The accident neurotic always dreams much and nearly always the dreams have a terrifying content. Thereby sleep is robbed of its refreshing effect and the patient wakes up weary and exhausted. Otherwise, too, *sleep* is disturbed, but usually superficially and for a short time. Where one can accurately determine the amount of sleep, as I, for instance, do regularly by the way I arrange the watching at night in my ward, one actually finds that the complaints of the patients in this matter are seldom exaggerated; by observation of persistent sleeplessness, I have learned to judge many accident patients mercifully, whose obtrusive lamentations and exaggerated complaints did not speak in their favor.

Among *physical phenomena* we find again the very same that we described under hysteria and neurasthenia (anæsthesias and hyperæsthesias, exaggeration of the reflexes, vaso-motor excitability, etc.). In this, I am inclined to see new proof of the close relation between hysteria and neurasthenia. I have seen in an endemic of accident patients that the same complaints and the same nervous phenomena, as, for instance, a marked acceleration of the pulse, recurred with remarkable uniformity, and only gradually became differentiated; I have seen in two patients narrowing of the visual field and isolated typical disturbances of sensibility. Are these cases to be separated from the others for this reason? I think that would scarcely be justifiable.

I can not take time here to go into the innumerable characteristic symptoms that some one or other thought he had found in the search for physical phenomena, only to recognize afterwards, that they are of no pathological importance. But there are three things I should like to mention, first, the *frequency of pulse acceleration*, which, as a rule, is noticeable even when the patient is sitting down quietly; generally flexing the knee a few times or rapid walking through the room suffice to drive the pulse to 120, 140, even to 160 and above. Occasionally pressure on a painful spot is also sufficient.

The second phenomenon is the *excretion of sugar*. One is often afforded an opportunity of seeing, after accidents, on one day a considerable amount of sugar, up to several per cent. which has absolutely disappeared by the next day. Other symptoms of diabetes are wanting. Even in cases, in which no sugar appears spontaneously, it is often possible to give rise to artificial excretion of sugar, a so-called *alimentary glycosuria*, by administering grape sugar. The ease with which metabolism may be disturbed is remarkable; we are not as yet in a position to interpret correctly this phenomenon, and the manner in which it takes place. But this does not prevent us from recognizing its importance as evidence of a rather serious illness.

And finally I should like to point out the importance of the *intolerance to alcohol*. We find, as is natural by collocation of the accident material, an extraordinarily great number of cases, in which the phenomena of *alcoholism* are closely interwoven with those of traumatic neurosis. Many a traumatic uses the time, so bountifully at his disposal, to drink perhaps even more than he did before. But in many cases the opposite takes place. They either give up alcohol entirely, because they observe the evil consequences of comparatively small quantities, now in the form of dizziness, or fainting spells or increased irritability to the point of violent fits of rage, now in lasting physical discomfort, now in the form of epileptoid states.

Differential Diagnosis.—I lay little value, as may readily be seen from what I have already said, on the distinguishing of traumatic neurosis from neurasthenia and hysteria; just as little on the assignment of the affection to one or another form of psychasthenic states. So far as differential diagnosis is concerned, the main things in question are chronic alcoholism, paralysis, and arteriosclerosis.

The frequency with which *alcoholism* appears simultaneously with the phenomena of accident neurosis, makes the decision, as to what is to be traced back to the accident, and what to drinking, difficult, especially since the intolerance to alcohol, which is to be regarded as a consequence of the accident, may appear as an additional difficulty. The one sided tendency of thought is far more conspicuous after accidents, than in the drinker, in whom, as a rule, one perceives a more general dullness. The mood of the alcoholist is generally cheerful, that of the traumatic, depressed. But often a long period of *observation in an institution* is necessary for a final determination of what has given rise to the condition of the patient. Symptoms of alcoholism disappear, except for small traces, in enforced total abstinence, unless a misuse of alcohol has preceded for years, which is then recognizable; the symptoms of traumatic neurosis, on the other hand, remain unchanged.

Since it has been proved, that *paralyses* may develop in connection with cranial injuries, one will have to investigate the previous history even more carefully than before, in order to determine whether the first traces of the disease do not go back beyond the accident. If this is the case, an important, the most important point is determined against traumatic neurosis. Otherwise the distinction is not easy, as long as the physical symptoms of paralysis are absent, or are so slight that they are still reconcilable with the symptoms that appear in traumatics, caused by slight innervation disturbances. The *writing* of the paralytic, which is an especially fine measure of the mental condition of many patients, furnishes the easiest possibility for recognizing the serious affection. Also in a *cytologic* examination we have a very good means of recognizing the organic disease, unless, owing to syphilis in the traumatic, an undesirable similarity of phenomena is again at hand.

Arteriosclerosis, as a rule, develops first at an advanced age; if it appears at the age of 40 and even before, and the symptoms of calcification of the arteries are very pronounced, the possibility grows that it is a consequence of an accident, and not a sign of physical involution. Why so frequently, and often in such an astonishingly short time, serious changes in arteries develop in connection with accidents, we are unable to determine; the fact, however, admits of no doubt. But thereby the difficulty is increased in distinguishing the consequences of an accident from natural involution at an advanced age. This can not be done by the symptoms alone. And one will usually be able to conclude, only from the fact, that the patient was able-bodied to the day of his accident, that the disturbances are to be traced back to the accident, that the arteriosclerosis in such cases is only a symptom of traumatic neurosis.

In the case of accident patients, one must not only decide whether accident neurosis is present, but also, to what *degree the ability to work and earn money* has been *impaired*. Here we are confronted with an almost insoluble problem. The valuation on a percentage basis of functional capacity can never be even approximately exact. With similar disturbances, the strenuous and ambitious man can do much, whereas the unenergetic man utterly collapses. The man who enjoys work will be able to accomplish something worth while, where the unwilling fellow gives out. And it is just this factor, *good will*, that one must take most into account as influencing, consciously and unconsciously, every attempt to work.

Unconsciously especially, in cases where the traumatic naturally lazy and lacking in energy, feels himself justified by his condition in considering himself unable to work. But not rarely there is added a *conscious* intention, not to use the remainder of his working ability, and by fighting for accident insurance to secure for himself a safe though small income. These conscious exaggerators and simulators are not always easily detected. Just because it is frequently a question of *psychic* phenomena, much may be simulated, without any one's being able to definitely prove simulation.

In my opinion, one can protect one's self from being deceived only by a very broad *knowledge of facts*. One must never forget that the proof of gross simulation of one symptom is no proof that all the other phenomena are simulated. This error in judgment is met but too frequently in expert opinions. Therefore I place little value on all the complicated methods, that are used to *expose* simulators. A *clinical observation* will always be the best road to the goal. An *unprejudiced one*, moreover, which neither aims at helping the injured man to secure unjust damages, nor is bent on exposing simulators, and, in proving the exaggeration, overlooks the really justifiable claims. I have always found that there was no more convenient, and with correct handling, no safer means, of distinguishing the disturbances actually present from those simulated and merely invented than meeting the patient with quiet earnestness, but without any mistrust. If he sees that one is not spying upon his every step, and not rejecting every complaint as unjustified, without further investigation, he will gain confidence; then it frequently happens that the patient at once confesses his exaggerations, or silently abandons them.

In the course of procedure I have just recommended, there is need of much experience and practice in the observation of psychic phenomena, otherwise all too great self-confidence might lead to equally important blunders, as too great distrust. I repeat again—traumatic neurosis is a *psychic affection*. The diagnosis of a psychic disease is conditioned not only upon the determination of various abnormal phenomena, but upon an exact knowledge of the *psychic personality as a whole*. This rule, applied also to traumatics, will make it possible, in the majority of cases, to really do the disease justice.

Prognosis and Therapy.—It is difficult to express the *prognosis* of traumatic neurosis in a few words. Too many different interests and chance factors are interwoven. A great deal depends upon, whether the patient from the first has had much opportunity for thinking about his troubles, or whether necessity has forced him to go back to work immediately. How much depends on the fact that the patient from the first has not looked upon his case as hopeless, I have already spoken of, in discussing the ætiology, and pointed to the importance also of the fight for insurance. Always, when it seemed to me possible, I have advised the patient to settle the matter as soon as he could, and rather effect an unfavorable compromise, than take upon himself the troubles of a long fight for the insurance money or of a law suit. The effect of *ending* a lawsuit by *compromise*, is often surprisingly good; it has been attributed by those who have little affection for traumatics to the fact that the patient, once his purpose is attained, has no ground for further simulation. This is very unjust. With the settlement of his claims to insurance, the main spring, or at least one of the most important of the troubles of the patient is stopped, and that he must improve then, goes without saying.

Theoretically, the most promising treatment is institutional—institutions in which the patient besides receiving a strengthening dietetic treatment learns how to *work* again. By systematic training in regular work under the guidance of the physician, it should be possible in the majority of cases to restore the patient to normal life. But, alas, these theoretically beautiful and hopeful attempts founder when face to face with the danger, that the grouping together of many such patients in the same house, brings with it. Here that querulous trait of character which I have already mentioned, has an exceptionally unfavorable effect, and a single element of that nature—and they are very plentiful indeed—can destroy the work of the physician, at one blow, at an entire sanatorium.

I should advise in every case that the very strongest and most suggestive assertions possible be impressed upon the patient of the certainty that he can recover. In all cases, in which the intelligence of the patient permits us to expect sufficient understanding, I point out the psychogenous nature of his troubles upon his own body and upon those of others, and urge him again and again to resume his work, for which the patient only gradually gets up his courage. But when he has once begun, he generally makes fairly satisfactory progress on the road to recovery. The attention of the patient must be again and again called to the fact, that even the largest income from insurance, is considerably smaller than the amount he can make as a healthy man and what he may make in addition by further advancement in his position.

In some cases I have also made use of *hypnotism*. It can only be used to get rid of some symptomatic individual troubles, especially those, which, like fear of riding on the cars after train accidents, are a hindrance in the patient's occupation. Sleeplessness and pains also may be beneficially treated by hypnotism. Occasionally by this procedure I have succeeded in getting rid of the fear of resuming work, even in cases in which treatment at home, in health resorts, in work sanatoriums, and private treatment had failed.

In cases of disease, which resist treatment, frequently a most unfortunate situation arises, a gradual sinking into more and more pronounced nervous conditions to such an extent, that, finally the patient exactly resembles the old cases of non-traumatic hysteria or neurasthenia. They seem to differ in one thing alone, that is, the tendency to *arteriosclerosis*. It cannot be denied that in isolated cases, even at an early age, arteriosclerotic changes appear very soon after accidents. At a somewhat more advanced age, the arteriosclerotic phenomena may finally, as we have pointed out, so predominate in the disease picture that one might think the case simply that of an ordinary arterial calcification if one had not seen the process develop under one's own eyes. Possibly—nearly always severe injuries are concerned—we may assume in these cases, that slight cerebral changes appeared at the accident. And I wish to emphasize that "possibly"; because we do not understand how it thereby happens, that the peripheral arteries also participate in this calcification process. So it is urgently recommended to be very careful in the prognosis, especially of older cases, with the assertion that complete recovery is possible, or to be expected—if not with the patient, at least with the insurance and accident companies.

5. IMPERATIVE THINKING

Definition.—The German word "*Zwangsvorstellungen*" (variously translated by obsessions, imperative or compulsory processes, ideas, thoughts, representations) was coined by *Krafft-Ebing*, who, however, understood thereby something different from the *disease* by which soon afterwards *Westphal* won his spurs in science. As a matter of fact, even before that time, many physicians, especially among the French, had long occupied themselves with what we now call imperative ideas or obsessions. *Westphal* wished to include under this head such ideas as, "when the intelligence is otherwise intact, and without being conditioned by the feelings or an emotional state, comes to the foreground of consciousness against and in spite of the will of the individual concerned; ideas that refuse to be dismissed and which hinder and interrupt the normal course of ideas, which the person himself always recognizes as abnormal and strange, and which, when his consciousness is intact, he resists."

The further development of the theory of imperative processes or obsessions, in which the French as well as many Germans participated, has furthered the knowledge of these states to an extraordinary degree. So large a number of forms has been described, each labeled with a name of its own, that pages might be filled with a mere mention of the descriptions. In contrast to these full clinical sources of information, stands the fact that many an important question is as yet unelucidated, that, indeed, the most important has remained unsettled, whether *Westphal's* definition was, or was not, correct. Thus three years ago in a critical review, *Bumke*, whose conception is intimately related to that of *Westphal*, stated the following as characteristic of imperative ideas: 1. The irremovability of the ideas and the subjective feeling of imperativeness that they call forth in the patients. 2. The absence of an emotional or affective condition, and 3. The retention of the critical faculty.

The form of obsessions which fulfills the three criteria of *Westphal* is doubtlessly of rare occurrence. If an idea always thrusts itself upon a sick person, as, why does the sun rise in the east, why is iron heavier than wood, why is glass transparent; or if he is compelled to utter a word, "worm, thunderstorm, etc.," every *emotional disturbance*, it is true, is absent, and then the patient feels such ideas to be abnormal, even when the questions are concerned with serious problems, as, who created the world. The compulsion lies in the fact that these ideas force themselves again and again into the consciousness of the patient, even when neither time nor place is suitable for such deliberations. But already in the very attempt to shake off these ideas, which in themselves have no emotional content—even exceedingly *troublesome feelings* and very gloomy moods may come if the patient seeks in vain to rid himself of these irrepressible ideas.

Still clearer is the emotional side in those otherwise slight cases, in which a patient, in passing along a street, is compelled to count or add the numbers of the houses. If he yields to this compulsion, the obsession does not disturb him seriously. But if he attempts to overcome it, there appears immediately an active *feeling of discomfort*, which may increase to an anxious, torturing restlessness, and force the patient, in spite of full insight into the idiocy of his actions, to return for several blocks to find the house-number that he had previously overlooked, or purposely left unnoticed.

I believe that we should no longer doubt, indeed should understand clearly, that an obsession is bound to be annoying by the mere fact that it intrudes again and again into consciousness, and must be unpleasant, because of the disturbances that it evokes; therefore, that an *emotional tone* is never absent. This would always be a *secondary* emotion, attaching itself to the obsession, whether to its content, or to the attempt to get rid of it or to thrust it aside. But even the simplest observation proves that very frequently the accompanying feeling of displeasure appears *synchronously* with the idea, and indeed frequently *precedes* the obsession.

If this is true of the lightest cases, it is still more true of by far the greatest number of serious forms, in which obsession makes its appearance. The patient, too, who during the discussion in the consulting room of the physician, calmly faces his obsessions, is able with complete critical *clearness* to show the insanity of them, immediately becomes the prey of occasionally very intense emotions, as soon as he attempts to *repress* his obsessions; and in the most severe forms, *anxiety* is finally the most prominent symptom, so that only an exact and careful investigation discloses the imperativeness of many conditions. It is these very cases, that give us a correct understanding of the nature of imperative thought. Under ætiology it will be stated more explicitly, that very frequent anxiety is the primary phenomenon, which the patient, so to say, attempts to explain by a certain idea, and the ever-recurring fear evokes the same idea. We may mention here, that even at *Westphal's* first lecture on obsessions, *Jastrowitz* pointed, in the discussion, with the utmost confidence, to the emotional disturbances as the origin of obsessions. Nevertheless, many German writers (*Hoche, Thomsen*) have clung to *Westphal's* conception up to the present day.

Just as little as I can recognize *Westphal's* first criterion as correct, so little can I assent to the second, that the *clear critical faculty* is always retained. I have been able to determine in a very large number of cases, that the patients, in the height of their terror, lost their critical faculty to a greater or less extent, only, I must confess, to recover complete understanding as soon as they were calm. If one has a chance to observe obsessions frequently, one will find countless times, that the patients again and again, while continually contradicting in great excitement, listen to the assurance that a red stain is not caused by sublimate, that a certain green does not come from verdigris, that it is impossible in passing to knock a man down and kill him without causing a disturbance, that a letter the content of which is exactly controlled contains no insult, etc., without being convinced. Everywhere, the critical faculty is somewhat, if not entirely, lost. It is characteristic, in my opinion, that the patient when resting, and especially in going over with the physician what has occurred, can see the folly of his ideas, only to lose his critical faculty again as soon as the obsession reappears.

My conception has, in the course of years, departed more and more from *Westphal's* definition, especially as I became more and more convinced of the fact that the critical faculty almost always suffers, and that the absence of all emotional excitement is very rare. Since the characteristic thing is not the content of the imperative idea, which is often formed by chance, I prefer to speak of *imperative thinking* and understand thereby *the appearance of ideas which, as a rule, are uniform, and mostly unpleasant, which—recognized inapposite by the patient when he is quite calm and using his critical faculty—impress themselves again and again upon the consciousness, associated with more or less strong feelings of displeasure.*

Symptomatology.—A division of obsessions (imperative thinking), according to content, leads to an endless splitting up into a thousand forms; it would be easy to add a new row of names to the countless names that have been invented for them. This seems to me absolutely futile. This disease is the same, whether the patient fears that he will become as red as blood upon entering a room, or fears that he has not turned off a gas jet, that he has made a mistake in counting, or that he has taken poison. I shall content myself, therefore, with describing some of the more frequently occurring types, which enable us to judge easily and correctly of the disease pictures that I have omitted.

The *least harmful* forms are those, in which the patient is forced to count objects, to add or multiply numbers, to hunt up names or citations, to recall to memory an absolutely indifferent word from a letter. But it may finally reach the point of driving the patient out of his bed in the middle of the night, in search of a quotation. In a few cases I have seen these same trivial obsessions lead to complete disturbance of every practical activity. One patient thought he was forced to hunt up every name that did not occur to him immediately, and write it down in order not to forget it. It gradually came to be for the most part geographical and historical names, for the finding of which he needed books of reference. But every time he opened an atlas or an historical work, new names appeared, and finally the patient occupied himself day and night with writing down names that he was afraid of forgetting.

A great number of obsessions arise from a feeling of *insecurity*, and I should even assert that in no sufferer from serious neurasthenia is this kind of obsessions altogether absent, even if it be only suggested. In this category, I place, for instance, the uncertainty, as to whether the gas jets are well turned off, the house door safely closed, letters properly signed and addressed, or placed in the right envelope, whether an account is correct, or orders for the kitchen and household already given. The uncertainty as to whether everything has happened, as he had proposed, may even come to the *healthy* man. He may, perhaps, return once to see. But the victim of an obsession, even after returning once, is not at rest. He knows that he has looked after it, but the fear makes him uncertain again, and finally he believes, that while he was looking after the things, he turned on the gas again, and opened the door already closed; now he must convince himself again, and then again and again, and so, in specially pronounced cases, according to my experience, the patient, the man servant and the maid move from room to room for several hours at night, in order to open and tightly close again and again all the doors, counting them loudly meanwhile. Or the patient opens a letter dozens of times, to prevent confusion, or waits for the emptying of the letter box, in order to convince himself again that the address is correct.

Here, too, in my opinion, belong all the cases, in which the patient does not dare to show himself in public. This is a torture, in the beginning, for many people, but the healthy person overcomes it and accustoms himself to speaking and appearing publicly, even though a remainder of discomfort and heart beating pursues many throughout life. This discomfort is distinguished from imperative thinking, however, in so far as the latter generally assumes a quite definite form. The most frequent kind is *stage fright*, taking the word in its widest sense. Though the patient knows exactly that he has mastered his rôle, he is overcome before his appearance by fear, which occasionally is of an undecided nature, but mostly attached to the thought that he might mispronounce an important word, lose his necktie at a decisive moment, have to sneeze, or something of the sort. One of my patients, a woman pianist, suffered from the fear that her hands might turn red while playing, so that she finally lacked the courage to appear publicly. A singer was unable to sleep for days before every performance, though she had never made a failure. Another singer wished to refuse, before his first appearance, under all sorts of pretexts, and could be induced to go on the stage only with the greatest difficulty. More frequently it is the fear of "getting stuck." The imperativeness lies in the fact that the patient convinces himself by repeating the piece he is to recite, a hundred times, that he actually does not "get stuck," and fully quieted rises from the piano or closes the book, only, a few minutes later, driven by the same fear to go over the passage again.

One of the subjectively most unpleasant forms is the *fear of blushing*. The patient believes that he will blush, as soon as he enters a room, or is seen. Frequently there is combined with this the fear, that one will suppose from his blushing, that he has thought of something improper, and so finally the idea becomes unbearable torture, which makes association with other people impossible. In the same way, the feeling of not being able to speak as smoothly as others may develop into an unbearable fear of stuttering, the fear of not being able to write well, into the impossibility of writing in the presence of others. Such patients know very well, that they can speak without stuttering, and write perfectly well. Especially when such a patient is with the physician, as a rule, not the slightest trace of nervousness in speech can be noticed, but as soon as he thinks that someone else might come, the fear appears that he will stutter then, or be unable to say a certain word. Likewise the fear of going to theaters and concerts develops. Such patients always sit in end seats, or in such seats that they can leave without exciting attention. They cannot go to a theater, or be in company, without the rising of such ideas, as "if you should feel bad," or "if you should faint." Occasionally, too, the content of the imperative thought is fear of the breaking out of a fire, or the dread of rousing the attention of the entire audience by speaking aloud or crying "hurrah." In vain, the patient seeks to shake off these fears, to explain to himself, that he never has fainted; he cannot get away from his fear. Finally such patients give up visits to theaters and concerts altogether.

Once I saw a case arise thus: The patient while sitting in a compartment of a train, in which there was no toilet, was seized with an attack of diarrhœa, but was able to reach the toilet in time, at the end of his journey. Thenceforth such an intense fear assailed him as he entered a train, that he was unable to ride in it, even when there was a toilet in his compartment.

The most characteristic and best known form of this sort of fear, is the *fear of open places.* In the moment, in which the sufferer from this form of obsession attempts to cross an open place or a rather broad street, he is assailed by a fear, occasionally with feelings of dizziness, which at times is quite vague, at times consisting of anxiety lest he become dizzy or faint. The patient usually knows how absurd this anxiety is. For instance, he says, that he can, without discomfort, worth mentioning, go through the open places, holding a two or three years old child by the hand, or that he could cross the street if only he touched with the end of his umbrella or cane the dress of a person walking in front of him. (One of my female patients, who thus attempted to help herself by touching with her hand, was in consequence suspected of being a pickpocket.) But as soon as the patients try to force themselves to cross the place without any assistance, a terrible fear overcomes them. They turn pale, begin to tremble, the sweat stands out on their foreheads, and they truly present a pitiable spectacle; the best proof of how strong emotional disturbance is in this kind of imperative thinking.

Fear does not always extend to what might happen to the patient himself. In many cases he is occupied by the thought that he has injured others. He has to pick up pins, because he is afraid that a child might swallow one. Every loose board evokes the thought in him that someone might fall over it, every match that a fire might be started. One of my patients gathered all orange peels, because a pregnant woman might slip, and be delivered prematurely; then she would be the cause of the death of the child. I have known a patient, who had the floor of a room torn up, in order to remove a match, which had fallen through a crack, though she could give very good reasons why such a match was harmless. An upper teacher got into difficulties in the course of the correction of the school exercise books, because the greenish shimmer of the red ink evoked in him the obsession that he might poison the children with verdigris. And although he knew that red ink made from eosin contained no copper, he did not get rid of his obsession.

The *fear of injuring others*, plays also the main rôle in patients, who are compelled to wash themselves all day long. They try, by as frequent washing as possible, to get rid of germs with which they may possibly have come in contact, of poison that may have come on their hands. And they wash themselves not only once, but several times, and then again and again in their anxious excitement, until half days go by in which they do nothing but wash themselves and cleanse the garments with which they have come into contact. A similar effect comes from obsessions of having touched something disgusting, and from that of having part of the Host on one's fingers. Such patients with the consciousness that in spite of all their efforts they will not succeed very soon in overcoming their obsessions, begin to dress hours before the proper time.

One of my patients came into conflict with her relatives, on a journey, because she never was ready in time for their engagements. No one suspected that she rose three or four hours before the others, in order to wash herself, and cleanse her clothes sufficiently.

In long standing cases of this sort, the patients finally come—one can scarcely describe it otherwise—to an *understanding with themselves*, not to wash more than say three or seven or twelve times, usually with the result that having reached the twelfth, doubt arises as to whether they have washed twelve times and they do not add one or two, but start anew on a series of twelve washings.

Now we are in the midst of *imperative actions*. It may be safely asserted that all imperative actions are dependent upon imperative ideas. Even such as, for instance, the compulsion to utter tic-like curses or obscene words, to touch every curbstone in the street in a definite rhythm, or in a restaurant, to choose the third table on the left side, at which to sit. One may say definitely that, almost without exception, the patient tries by such action to make some sort of a defense against his imperative ideas or attempts to escape their constant annoyance.

To imperative processes belong also the *imperative impulses*. Harmless ones, as suddenly rising in the midst of a concert, suddenly crying hurrah in a theater, spitting down from above, taking off a stranger's hat on the street, and others of the same sort. Far more annoying are the thoughts of placing stones on the car tracks, of striking children or pushing them down a flight of stairs, of insulting harmless passers by with abusive words, or even terrifying them by obscene gestures. According to my experience, all these imperative impulses are never transformed into imperative actions, in so far as they are not of a harmless character, or unless they can be carried out in such a way as to rob them of all possible danger. Thus one of my patients always used the opportunity, when a wagon drove by with much noise, to quickly utter an oath, but could always control herself, when she could not employ this method of ridding herself of her imperative impulses.

Many other actions of a harmless nature, as washing, closing the doors, picking up pins, I have already mentioned. The patient may by yielding to his obsession, be absolutely prevented from doing any work, may thereby endanger his social position, but will never permit himself to be carried away into an action, that might become dangerous for another. But in every case in which under the influence of imperative impulses actions of a questionable or even of a criminal nature were committed, one is not mistaken in assuming that a serious affection is concerned, which may no longer be reckoned as belonging to the realm of imperative thinking.

Etiology.—I have purposely placed the discussion of the causes of obsessions (imperative thinking) at the end of these descriptions, because much is thereby, without further elaboration, made intelligible. I shall attempt to explain by a few more examples, how obsessions may originate. A gentleman had to give his signature and thereby his assent, in a large assembly, to an undertaking on the outcome of which his entire social position depended. A by-stander mocked, as he perceived how the man trembled in writing his signature. Thereafter, as often as he had to write his name in the presence of others, the idea appeared, that he had to tremble and, finally, he was absolutely unable to write in the presence of others. Another man saw the lightning, in a storm, destroy the chimney of his factory. Since that time, he has lived all summer in constant fear of every storm and always was compelled to think that the lightning would strike the chimney of his factory again, whereas he was altogether indifferent to the possibility that the lightning might strike his own or some one else's home. A particularly obvious example, is that of the female patient already mentioned who could no longer ride on the train.

These examples point to one possibility of how imperative ideas develop. But we are neither able to prove similar causes for each case, nor justified in seeing therein the nucleus for the development of the disease. Countless persons suffer some terrible mischance, which perhaps leaves discomfort behind it for a long time, as soon as a similar situation or any other chance evokes the memory of the occurrence, but it reaches imperative thinking only in persons specially predisposed to it. Therefore I consider it very important to speak, in general, not of imperative ideas, but of imperative thinking.

The justification for this lies in the experience, that many patients have various kinds of imperative ideas, e. g., fear of open places and dread of not having shut the door, whereas, at the same time, they are perfectly free from fear of touch. Occasionally, we see one kind of imperative ideas disappear, only to be replaced by another.

Thus in one of my female patients, as soon as she heard of the illness of even a distant connection, the thought forced itself upon her what would happen in case of death, which of the relatives would be the most excited, etc., and alternated with the imperative idea that she must blush in company. In another, the compulsion, find the square of all numbers alternated with the fear that she might make other people sick, by imagining that they were affected by illness. One of my patients suffered now from fear of open places, now from the imperative idea of being compelled to disturb public assemblies by loud cries. In all the cases, where the content of the ideas changes, the patient wonders, while occupied with one, how he could ever have taken the other so seriously, and he is just as regularly astonished if one describes to him the imperative thinking of others, at the strangeness of the extraordinary idea, and thinks that the imperative ideas of others can certainly be easily suppressed.

These experiences point to the fact, that in the causes just mentioned, we have nothing else before us, than the process, that gives the imperative thinking its direction, the imperative idea its form, but back of all, we recognize the constitutional foundation which, on the whole, resembles that of the neurasthenic.

Differential Diagnosis.—As a rule, the *diagnosis* of imperative ideas causes no difficulties. The manner in which the patients present their troubles is so characteristic, that they could scarcely be confused with anything else. Some abnormal conditions in cases of dementia præcox have an external resemblance with imperative ideas, but in these cases the patients lack the feeling of the necessity of thinking, which intrudes itself imperatively, and the patients do not suffer so much from their troubles as those affected with imperative ideas.

Occasionally, *manic depressive insanity* and especially *periodic depression* appear in the form of imperative ideas. The regularity, with which this illness repeats itself, distinguishes it from the constitutional state of the sufferer from imperative thinking.

Prognosis and Course.—Obsessions appear occasionally at an extraordinarily early age. The further development depends not a little on the treatment, as is best disclosed possibly by the fact that among the working people obsessions virtually never appear. Obviously for the development of this disease, there is need of time to devote to brooding, in which to occupy one's self so long with the recurring ideas, to worm one's self so thoroughly into them, that finally imperative thinking is stronger than the energy of the patient.

Social utility can be seriously affected by imperative thinking, as I have already explained. It is natural, that the simple mechanical hindrance of a person who suffers from fear of open places or of one who has to test everything over and over again, the fear of touching unclean objects, or however else the content of the imperative thought may appear, finally cuts off the patient, so to say, from intercourse. In spite of that I do not think the prognosis so unfavorable as is often supposed. I have seen, too, frequently good results from treatment, though I must confess that treatment can be effective, only when it is continued for a sufficient length of time, and that the tendency of new obsessions to appear, persists under unfavorable conditions.

Treatment.—Imperative thinking, as a constitutional disease, is susceptible to treatment only to a limited degree, if a *cure* is regarded as the aim. On the other hand, a great deal can be attained, if one is satisfied with getting rid of the most painful obsessions of the patient, and perhaps passing beyond that, attempts to teach him how in the future he can prevent his doubts and anxious ideas from becoming fixed.

Every exhausting influence strengthens imperative thinking, and therefore the treatment must frequently begin with the recuperation of physical forces. Furthermore, every *occupation*, especially manual labor, is of useful assistance, if it is not a means of cure. On the other hand, little can be expected from medicines, though it may be quite pleasant to the patient, to be able to quiet himself by a few valerian drops. The main thing will always be the *psychic influence*, and this can not be properly directed too soon or with too much energy.

No scheme of combating the individual obsessions can be given. Every idea must be grasped in an individual way, every patient treated according to his peculiarity. At times, the best policy is to permit the patient to yield utterly to his fear. The result of this may be that he is less afraid of his obsessions, and, therefore, free from strong emotions, more easily sees their absurdity. But this method is only rarely permissible. For the most part one will have to try to draw the patient *slowly* and gradually away from his ideas, to explain to him that the content of his imperative thinking is an affair of chance, since he is accustomed to estimate correctly the absurdity of the obsessions of other people. In this very process of shaking his faith in the sanity of his own ideas, of getting rid of the concomitant or preceding emotions, I have used *hypnotism*, with good results. In view of the opposition that is always shown to this method of treatment (cf. Page 909), I consider it necessary to emphasize the fact, that by means of hypnotism, many a patient suffering from obsessions has been made over into a useful man, who for years had in vain gone from sanatorium to sanatorium, from physician to physician.

Ceaseless work, the undertaking of serious duties, are indispensable helps in treatment, which but rarely attain their goal in a short time. It is mostly an endless wearisome wrestling with one persistent fast-rooted idea, often also a fight, in which for every fear that is gotten rid of, another immediately appears, until finally the patience of the physician and the endurance of the patient win the victory, and this victory is worth while, for few patients suffer so much as those who are tortured by obsessions, and, at the same time, few find so little understanding in those about them.

6. MIGRAINE

Definition.—By *migraine* we mean attacks of *pains in the head*, which affect *one side of the head* with special severity, and are usually accompanied by *nausea* with vomiting and *disturbances in sight*. As a rule, other nervous disturbances are present, so that the migraine is not only a symptom, but an extraordinarily obstinate and painful one, of general psychopathy.

Only in a few diseases do we find the fact hold good as often as in migraine, that in the *family*, in parents, sisters, brothers, or children the same phenomenon appears, and there are whole families, in which scarcely one member is free from a predisposition to migraine.

Only in late years has great emphasis been laid on the relationship between migraine and *epilepsy*. That they are closely related can scarcely be doubted, if one considers the frequency with which, in one family, some of the members suffer from epilepsy, and others from migraine. If one adds the experience, that often individuals who are undoubtedly epileptics show isolated attacks of migraine, that at times attacks of migraine appear for years, and later give place to typical epileptic seizures, or that migraine-like pains in the head appear as initial symptom of a serious epileptic attack, and if one further considers the relations of *alcohol* to migraine and epilepsy (which are to be discussed later) definite conclusions may be drawn from this as to the very close relationship between the two diseases. Whether one is justified in regarding every attack of migraine as an *epileptic equivalent*, must, however, appear doubtful. We see too often the combination of migraine with other neuroses; and also from the effectiveness of treatment may be drawn the conclusion that migraine is not always to be regarded as epileptic.

Women are somewhat more frequently attacked than men. But the differences are not very pronounced, and possibly explicable by the frequency with which the attacks of pains in one side of the head are related to *menstruation*. A great many women suffer from attacks of migraine just before or at the end of the menses, while they are absolutely free from them at all other times. Occasionally one observes migraine conditions during *pregnancy*, which after the birth cease completely until the next pregnancy.

The first attacks happen usually during the *period of development;* it is relatively rare that they appear for the first time at an advanced age. As external causes, overexertion, and, not infrequently, physical disease may be mentioned. Whether migraine can actually arise from *external irritations*, disturbances in the nose, etc., I think doubtful. On the other hand, it is quite certain that with an existing predisposition to migraine, the single attack may be brought on by external irritations, and its appearance prevented by avoiding them. The patients, as a rule, know very well what is injurious to them, and seek, each according to his or her disposition, to avoid bright light, irritations, bad and smoky air, digestive disturbances and constipation.

Especially important as a cause of the isolated attack, is alcohol. Every indulgence in comparatively large quantities, as, for instance, half a bottle of wine, is sure to give rise to an attack; in many people, the small quantity involved in a very modest, but regular daily indulgence, suffices to bring on migraine from time to time, as the absence of attacks during total abstinence proves.

Symptomatology.—As a rule, the attack is preceded by a feeling of *weariness* and exhaustion. The patient feels tired, unable to do anything, his limbs are heavy, his head benumbed. The onset corresponds somewhat to conditions such as appear in the incubation stage of serious infectious diseases. Occasionally, disturbances of the *digestive tract* appear before the *headache.* Then the headache soon begins, and, as its name denotes, is confined as a rule to one side of the head. But this restriction is true only of the beginning of the attack; when it has lasted for some time, the pains gradually radiate to the other side, or shoot, though infrequently, over to the other side, so that the side originally affected, is quite free from pain. Many patients have the attacks on the same side, all their lives, whereas in many others, the attack is sometimes on the right, sometimes on the left side.

Though pains may occur in the *back of the head*, the pain, as a rule, is in the region of the temples. It is now piercing, now stinging, now dull, but always extremely violent. During the attack, the patient is hypersensitive to external irritations, especially to light and sound. But even cold irritations (draughts) make the attack worse.

Only infrequently is *nausea* absent, nausea which may increase to *vomiting.* Vomiting only temporarily relieves the feeling of nausea, usually the act of vomiting actually increases the headache. Occasionally *diarrhœa* appears, but is often absent. The patients complain of *cold*, *shivering*, less frequently of being hot.

Almost regularly, at the beginning of the attack, appear symptoms that are concerned with the eyes. In many patients, the eye symptoms usher in a series of serious disturbances. Usually the patients complain that they are suddenly unable to see with one eye—oddly enough it is sometimes the eye not corresponding to the seat of the migraine—or that a dark spot appears in the field of vision. The spot is absolutely dark, or gray, or surrounded by brilliant colors. A characteristic, if not regular phenomenon in migraine, is the appearance of a scintillating *zigzag* figure: parallel lines *running in zigzag fashion* passing to and fro before the eyes, the so-called *Flimmerskotom.* At the height of the attack, as a rule, the disturbances of the eyes disappear or lose their troublesome nature.

During the attack itself, the *pupils* are frequently contracted, which is the more important as the patients at that time prefer to stay in the dark. But the testing of the pupils is very difficult, since the supersensitiveness of the patients to light makes the opening of the eyes far from easy, and closing the eyes tight, *per se*, may of course cause contraction of the pupils.

In serious cases occasionally physical phenomena appear, which might cause even the suspicion of *hemiplegia*, if experience did not force upon the patient himself the conviction, that it was only the usual attack of migraine; feelings of weakness, which may be combined with all possible unpleasant sensations in arms and legs, at times even genuine *aphasic disturbances.* At any rate, it is difficult to decide whether the patient actually is unable to find the word, or merely finds difficulty in recollecting it, because of the violent pains and preoccupation.

Occasionally at the climax of specially serious attacks *psychic disturbances* appear; the patients are irritable and fly into fits of rage, or begin to complain, fear a stroke or insanity; it may even increase to pronounced *confusional states*, which in their peculiar brutal irritability, their difficulty in recollection, the absence of or slight clearness of later remembrance precisely recall epileptic fits. In these cases, too, the relationship to epilepsy already mentioned again confronts us, and in cases where later migraine disappears, and in its stead typical epileptic fits, either with or without the accompaniment of one sided headache develop, I see as a matter of fact no possibility of regarding the first unilateral headache as anything other than an epileptic equivalent.

The **diagnosis** of migraine is, on the whole, not difficult. Confusion with *tumors of the brain* is possible, in them, however, the headaches rarely appear on one side only, and more rarely, at regular intervals. Occasionally —and this is important for making the diagnosis—migraine, and especially the form with decided eye symptoms, appears at the beginning of progressive *paralysis*. The more advanced age, at which the first attacks come, points from the beginning to a serious organic disease, which, as a rule, then betrays itself by other physical symptoms. In every case it is advisable to pay special attention to the general constitution of the patient, since, for example, the migraine of the hysteric is far more susceptible to treatment than is the idiopathic form.

Course and Prognosis.—Migraine belongs to the most stubborn diseases that we know, and many patients wage an unsuccessful war with their torturing affection for years. In women, improvement comes occasionally at the time of the *climacterium;* in men at a more advanced age. But this comforting thought that one is so glad to be able to offer patients is, alas, not always justified. Rare, in spite of the violent suffering, is a serious injury to health, other than the fact that the patient becomes run down from frequent attacks, and other than in the cases in which epilepsy appears as an initial symptom of paralysis.

Treatment.—During the attack, the patient desires nothing so ardently, as *rest;* most patients lie down in as quiet a room as possible with darkened windows, to wait for the end of the attack. Many are in such need of rest that they oppose every means that one tries upon them, among which I include hot baths 98-104° F. of short duration (2–3 minutes), hot foot baths with the addition of mustard; with other patients immersion of the feet in hot water, and occasionally of the hands also is effective. As a rule, the patient has tried everything and knows what gives him some relief.

Medicines can scarcely be dispensed with in any case. Often a remedy works excellently in one attack, only to fail in the next, or it alleviates the troubles for months, only to be absolutely ineffectual afterwards. As a rule, one ought not to work with too small doses: *antipyrin* 1.0, *phenacetin* 1.0, *caffein* 0.15 to 0.2, *trigemin* 0.75, *pyramidon* 0.3, are some of the most useful remedies in such doses as one usually prescribes for an otherwise healthy man. In the beginning of the attack I have frequently seen good effects from a cup of strong *coffee.* In many cases *bromine* (5–6 g.) works excellently where all other remedies fail. I must emphatically warn against the use of *morphine.* Because of the regularity with which attacks usually recur, the danger of *morphinism* is so great that up to the present I have not been able to make up my mind to use morphine in a single case. Nor have I seen much good resulting from the morphine in the cases in which it was prescribed upon the authority of others. If the attack lasts beyond a day, occasionally soporifics in not too small doses (trional 1.5, vernal 0.75) are useful.

It is appropriate to treat the sufferer from migraine before the attack starts. Especially in the cases in which association with epilepsy is suspected may the number and severity of the attacks be reduced by long continued daily doses of 2–3 g. bromide. In these cases the assistance afforded by *total abstinence from alcohol* is of extraordinary importance. But in all other sufferers from migraine as well, total abstinence from all forms of alcoholic indulgence is one of the most important means of improving the disease. General dietetic measures (restriction as to diet, care as to the regular evacuation of the bowels, etc.), hydrotherapy in mild forms, and finally preparations of iron and arsenic are useful means of removing the existing nervous tendency.

IX

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM

BY

L. R. MUELLER (Augsburg)

A presentation of the pathology of the sympathetic nervous system is much hampered by the fact that there is still much uncertainty as to its physiology. In the course of the last years, it is true, English physiologists, especially *Langley* and his school, have undertaken many investigations dealing with the autonomous nervous system in different animals, but we must confess, that we still lack a real understanding of the innervations here concerned. The relations are quite different from those in the cerebro-spinal system, in which certain symptoms of functional loss follow and correspond to the focal diseases. Breaks in the conductivity of peripheral nerves lead to definite motor paralyses and to restricted sensory disturbances. It is not so in the autonomous nervous system. In lesions of the sympathetic ganglia, or after complete division of their peripheral branches, the results are at most temporary disturbances, but not complete loss of the functions of the organs concerned.

Thus the heart, even when severed from all nerves, works on in a way fully sufficient for the maintenance of life; thus, the peristaltic action of the bowels is not paralyzed, even after complete division of the splanchnics; even a womb cut off from all the pelvic nerves, can powerfully eject its fruit at the proper time.

The scheme that we drew up of the nervous processes in the cerebro-spinal system absolutely fails in any attempt to explain thereby the physiology of the sympathetic. In fact, every attempt at a scheme inevitably founders upon the rocks of anatomical relations. In the richly branching nerve plexuses, such as supply the heart, stomach, intestines, liver, kidneys, the larger vessels and the pelvic organs it is impossible for the anatomist, as well as for the physiologist, to find his way, and to ascertain what functions belong to one or another of the fibres.

Thus the vagus and sympathetic, even during their course in the neck, enter into numerous connections with each other. The plexuses they form together in the heart and the abdominal cavity can not be disentangled;

nor is one in a position to decide as to what extent the fibres in the pudendal plexus spring from the cerebro-spinal and to what extent from the autonomous systems. These plexuses, moreover, and even the location of the sympathetic ganglia, are subject to great individual differences.

Before discussing the pathology of the autonomous nervous system, it may be advantageous to recapitulate briefly *what we think we know about the course of the sympathetic fibres from their origin in the cerebro-spinal system to their end in an organ.*

Immediately after the anterior roots have joined the posterior roots that have penetrated the spinal ganglion, to form the spinal nerve, there spring from the latter, the white rami communicantes (cf. Fig. 218). Their small medullated fibres spring for the most part, unquestionably, from the anterior roots and, indeed, there is reason for supposing that the appertaining ganglionic cells are situated in the lateral horns of the gray matter and in the vicinity of the central canal.

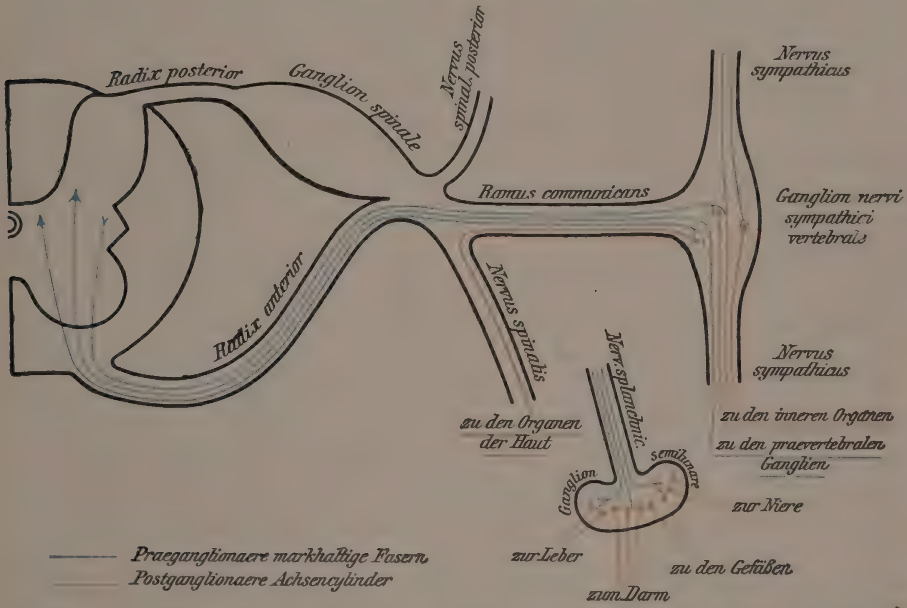

FIG. 218.—Scheme of the efferent fibres of the sympathetic nervous system. Blue, pregangliar medullary fibres. Red, postgangliar axis cylinders. In the cut, *zu den Organen der Haut*, to the cutaneous organs; *zur Leber*, to the liver; *zum Darm*, to the intestines; *zu den Gefaessen*, to the vessels; *zur Niere*, to the kidneys; *zu den preavertebralen Ganglien*, to the prevertebral ganglia; *zu den inneren Organen*, to the internal organs.

The rami communicantes turn, as single or double nerve bundles, to the next ganglion of the gangliated cord. In this ends the first neuron, which *Langley* designates as "preganglionic" and enters into contact through and end arborization with the second, the "postganglionic." The postganglionic nerve fibres have no medullated sheath; they turn either to the organs that are to be innervated by them within the trunk, or they reach the spinal nerve again, by way of the gray rami communicantes, which mostly course in the same nerve bundles as the white ones, to pass with its main branches to the organs of the integument, the vessels, the sweat glands and the unstriped muscles of the hair follicles (cf. Fig 218). But in many cases the fibres of the white rami communicantes pass through the next ganglion, without any interruption, and only unite with the second neuron in a more viscerally situated, so-called prevertebral ganglion. Part of the rami communicantes unite to form larger white trunks (pars cervicalis nervi sympathici; nervi splanchnici) and thus reach comprehensive prevertebral ganglion formations (ganglion stellatum, ganglion cervicale superius, ganglion cœliacum), from which then spring the non-medullated, postganglionic fibres for the vessels and the inner organs.

In this connection it should be recalled that to-day the sympathetic nervous system does not imply only the bordering cord running along the spinal column, with its ganglia and extensions, but that to it belong also the fibres that leave the cerebral nerves and pass to the ganglion ciliare, spheno-palatinum, oticum, submaxillare and sublinguale, as well as fibres which spring from the sacral nerves, and lose themselves in the ganglia and plexuses of the true pelvis (autonomous bulbar and sacral system).

The *innervation territory of the vegetative nervous system* extends to all the glands in the body, wherever they may be situated, and to all organs with *unstriped musculature.* By the sending off of preganglionic fibres from the cerebral nerves are innervated also the glands in the head, the lachrymal glands, the salivary glands, and the mucous glands in the nose.

Irritation of the cervical sympathetic is followed by dilatation of the pupil (contraction of the dilatator pupillæ) and protrusion of the eyeball from the orbital cavity (contraction of *Müller's* muscle); there is, moreover, a contraction of the vessels of the face, the conjunctiva and the retina. From the cervical sympathetic delicate plexuses also enter with the large vessels the cranial cavity. Irritation of the vasomotor fibres, or heightening of their tone, is followed here, as in the trunk and extremities, by contraction of the blood-vessels. Whether in addition to the vaso-constrictors, there are also vasodilatator nerve fibres, has not been definitely proved, but a number of physiological experiments and some physical phenomena (erectio membri) speak for it.

It is not always easy to decide how far the activity of the glands, as for instance, of the salivary glands, or of those of the liver or the kidneys, is indirectly stimulated by vasomotor processes, and how far the sympathetic fibres influence secretion directly. "Cold sweat," i. e., the production of perspiration without hyperæmia of the skin, the secretion of gastric juice due to sensations of smell and sight, and several other observations permit us to conclude with certainty, that the preparation and emission of secretions can be directly stimulated through sympathetic paths. On the other hand, one must point out the dependence of glandular activity upon *local* stimulations. Thus local heating of the skin causes hyperæmia and the secretion of perspiration. Upon stimulation of the mucous membrane of the mouth, salivary secretion takes place, and upon contact of the mucosa of the duodenum with hydrochloric acid, there is a secretion of the digestive glands.

As are all glands, so also are the glands and unstriped musculature of the bronchial tubes innervated by the sympathetic system; hence attacks of serious bronchial and nervous asthma after mental excitement speak for the dependence of the sympathetic innervation upon psychic emotion.

The autonomous system—and to this belongs the vagus after its entrance into the thoracic cavity—exercises only an accelerating or retarding influence upon the action of the *heart* and the peristaltic movements of the *intestinal canal. The impulse to work lies in these organs themselves.* It is well known that the vagus branches have a retarding, while most of the sympathetic fibres have an accelerating effect on the heart beats. On the other hand, the sympathetic seems to exert an inhibitive influence on the peristaltic movements, through the splanchnics, but the observations on the subject are, as yet, contradictory.

The innervation relations of the *bladder*, *rectum*, and the *sexual organs* are especially difficult to determine. To understand these even partly, one must perceive clearly that here too the actual seat of the innervation is to be sought in the organs themselves, and in the sympathetic plexuses situated in and about them. By striped sphincter muscles, which can be influenced voluntarily, the receptacles of the urine and the feces are shut from without, and by their help are suppressed inopportune peristaltic movements of the unstriped musculature of the bladder and the ampulla recti.

On the other hand, we are in the position by voluntary innervations, as for instance, by the use of abdominal pressure, to excite the automatism of the movements which lead to evacuation of the feces or the urine, and by certain sensory stimulations to excite the complicated process of emptying the sexual glands.

On the contrary, the activity of the other organs that are supplied by the sympathetic, as the heart, intestinal canal, the vasomotors, and the glands, can *not* be influenced *voluntarily* at all, and yet the autonomous nervous system is connected with the cerebro-spinal by the rami communicantes. Here it must be pointed out that not the will, but *moods* and *emotions* and *inclinations* may work upon the function of the organs mentioned. Thus in terror the blood-vessels contract, the face pales in anxiety, and in fear there is an outbreak of perspiration or a contraction of the erectores pilorum (goose flesh); indeed, as a consequence of the contraction of *Mueller's* muscles, the eyes may fairly bulge from a face distorted by terror. With joyous moods the cheeks redden, the heart beats more quickly, it "jumps for joy." Psychic pain, on the other hand, leads to secretion of the lachrymal glands.

The influence of psychic processes on the internal organs may be increased to such an extent as to become really pathological; thus, in some nervously constituted individuals, after psychic excitement, whether agreeable or disagreeable, there is vomiting, in others, with the same causes, there are attacks of diarrhœa (emotional diarrhœas) or dysuria.

The question, by what paths in the brain and spinal cord this influence is mediated, can not in the present state of our knowledge, be answered. In the first place, one would have to consider whether definite groups of fibres are not reserved in the spinal cord for each individual function, as for the vasomotors, the piloerectors, the heart, stomach, intestines, and the various glands. But spinal-cord pathology does not furnish us material to corroborate this point of view. The single columns of the spinal cord are all known, with respect to their kind and their function, and nowhere can there be localized on a cross-section of the spinal cord, such numerous fibres as would be necessary for the acceptance of this assumption.

Now if no special fibre system in the brain and in the spinal cord can be made responsible for the influence that emotional disturbances have on the activity of the internal organs, there may be a possibility that there is no need for special paths, and that by the emotions, the nervous excitability as a whole, is in a typical way, "tuned," i. e., the origin cells of the rami communicantes would then react in their way, as percipient organs upon the quantitative or qualitative change of animal electricity in the cerebro-spinal system. That, under the influence of emotions, there is actually a change of the innervational relations, may be deduced from the fact that sensations of pleasure tend to facilitate, while sensations of displeasure tend to retard the regular course of the innervation. The influence upon the sympathetic and psychomotor nervous systems at one and the same time, is best exemplified by terror. This conditions not only the constrictory function of the vasomotors, a retreat of the blood from the surface of the skin, acceleration of the heart beat, stimulation of the piloerectors, but can also "make us weak in the knees" and "paralyze" the limbs, or make them shiver and shake.

Heretofore, only the *efferent* paths of the autonomous system have been discussed; whether, besides these, in the sympathetic there are also *afferent*, i. e., centripetal ones, is a moot point, answered in the negative by prominent anatomists and physiologists (*Koelliker*, *Langley*). It is certain that the cervical sympathetic and the sympathetic fibres, which course with the peripheral nerves to the organs of the skin, have no sensory fibres.

Recently, surgeons also (*Lennander*, *Wilms*, and others) have said, on the strength of their operative experience, that there is no feeling in the organs innervated by the sympathetic, and that the pains that we locate in the abdominal organs, are always caused by irritation of the parietal layer of the peritoneum, which is indeed richly supplied with spinal fibres. On the other hand, we must point out that the ganglia of the bordering cords have a histological structure wholly similar to that of the sensory spinal ganglia. It has even been conjectured from an embryological point of view that the former undergo segmentation from the latter. Also many physiological experiments and clinical facts, make probable the existence of centripetal fibres in the sympathetic system. Thus the pains in angina pectoris, the gastralgia in increased secretion of hydrochloric acid, the painful colics in the expulsion of gallstones or renal calculi, can not, without stretching a point, be explained by the irritation of the parietal serosa. It is true that the sympathetic paths which convey pain impressions from the internal organs centripetally apparently have no direct ascending continuations in the spinal cord and no peculiar projection field in the brain. On the contrary, it has been proved by the investigations of *Head*, that in diseases of the internal organs, pains frequently appear in those cutaneous zones, the spinal fibres of which pass into the same spinal segment as the corresponding rami communicantes, nor do *spontaneous* pains only appear there, for the cutaneous zone concerned is also strongly hyperæsthetic to slight pain stimuli. Investigation reveals the existence of such hyperalgetic sections of the skin, in the majority of cases of stenocardia, ulcers of the stomach, cholelithiasis and nephrolithiasis, indeed also frequently in kidney diseases. In the spinal cord there seem to be close relations between the sensory nerves of an organ and the sensory cutaneous nerves. The close contact between the two conducting paths can take place only in the gray matter of the posterior horns. For it has been proved in the first place, that all pain-bearing impressions, which come from the outer integument, are conducted through the posterior horns; on the other hand, there are reasonable grounds for the assumption that the afferent fibres pass through the posterior roots. Thus it is comprehensible that there exists only a *hyperalgesia* but not a general æsthesia of the cutaneous zones concerned. Since the sympathetic paths have no direct continuation in the spinal cord, but can apparently give rise to a conscious sensation only by irradiation through the fibres from the outer integuments, it is really no wonder that the localization of pains in organic diseases is less sharply defined.

In the *pathology of the autonomous nervous system*, there is only one clearly marked clinical picture, namely, the paralysis of the cervical sympathetic. This appears after formation of tumors (struma, lymphoma, aneurisms), especially after traumas (injuries through stabbing or shooting), and after surgical operations on the throat. The symptoms of complete division of the cervical sympathetic are: contraction of the pupil on the same side as a result of paralysis of the dilatator pupillæ, diminution of the palpebral fissure, and retraction of the eye-ball, as the result of paralysis of Mueller's muscle, hyperæmia of the skin of the face and the ears, as a result of paralysis of the vasoconstrictors concerned, and anhidrosis. But the fact must be emphasized that all the disturbances mentioned may disappear in the course of a few weeks. Contraction of the pupil persists most obstinately, but never leads to loss of the light reflex. Growing tumors, like malignant enlargements of the thyroid gland, not seldom lead at first to an irritation of the cervical sympathetic, which causes dilatation of the pupil, exophthalmus, pallor of the face, and hyperhidrosis; and only later with the development of pressure atrophy, do the above-named phenomena appear.

In the thoracic and abdominal cavities, the bordering cords (sympathetic nerve) with their ganglia and plexuses, lie so sheltered that traumatic lesion can scarcely ever occur. But we know nothing positive even about the influence of tumor formations in the spinal column, upon sympathetic fibres there. Such an influence may possibly exist there, in slight degree, and temporarily, but because of the great independence of the organic functions, it can never result in severe disturbances.

Just as little do *focal diseases* of the spinal cord cause pronounced phenomena of irritation or loss of function in the region of the visceral nervous system. Well known are the disturbances of the pupillary innervation (mostly miosis) in focal lesions in the lowest cervical and upper dorsal portion of the spinal cord, and slight vasomotor disturbances (paralysis of the vasoconstrictors with cyanotic coloring of the cutaneous segment affected) in poliomyelitis and syringomyelia. The activity of the heart, of the stomach and intestinal canal, of the liver and the kidneys, is not influenced by local spinal diseases. On the other hand, the excitation of the reflex which leads to the discharge of feces and urine, may be made difficult, or even entirely prevented by transverse affections or by lesions in the lowest segment of the spinal cord.

Of the systemic diseases of the spinal cord, *tabes* almost as a rule, is accompanied by disturbances in the autonomous nervous system. How far miosis and the reflex pupillary immobility to light are to be connected with degeneration of sympathetic fibres and with that of the fibres of the cerebrospinal system, it is difficult to judge. On the other hand, the gastric and intestinal crises, the bladder and rectum disturbances, the impairment of potency and the tachycardia, so often established in cases of tabes are unquestionably to be traced back to diseases of vegetative paths. Where, indeed, this disease is to be localized, whether we must seek for the cause of visceral crises in the rami communicantes and their continuations in the spinal-cord roots, or in the ganglia of the bordering cords, and in the postganglionic neurons, we have no sources of knowledge.

The *functional* disturbances of the sympathetic nervous system play a large rôle in neurology. It is the vasomotors above all to which the complaints refer. Unilateral reddening of the face combined with a feeling of great heat, sudden macular reddening on the neck and breast, and on the other hand, facial pallor with a cadaverous pallor of the nose and the ear are frequent troubles. If the vasoconstriction is very strong, as in "dead fingers," there may be unpleasant sensations, as formication, a feeling of the parts "having fallen asleep" and even pain. Such troubles may appear in individuals usually quite healthy, but the neuropathically predisposed, like neurasthenics and hysterics, are especially inclined thereto. With the appearance of the menopause there come to almost all women vasomotor disturbances, the so-called heat flashes. Let us point out very briefly that the vessels, as a result of nervous influences, may, under certain conditions, come to be more pervious than normally to the blood serum, and that then more or less extensive swellings of the skin (urticaria or acute angioneurotic edema), or diffusions into the joints (hydrops articulorum intermittens) can arise. One must mention, furthermore, that migraine is also to be counted among the vasomotor neuroses. According to our present conception, its fundamental disturbances are occasioned by the vasoconstriction of central vessels. Ischæmia of the heart-muscles, appearing in paroxysmal attacks, leads to peculiarly painful and annoying conditions. Such conditions of "angina pectoris" appear in people in whose arteries one sees signs of old age, often associated with psychic excitement; they are mostly associated also with vasoconstriction of the facial vessels, with great facial pallor.

The anomalies of *sweat secretion* are relatively unimportant. Occasionally there is increased production of sweat in one-half of the face, or of the body (hyperhidrosis unilateralis). One speaks of hyperhidrosis plantaris and palmaris, if upon the soles of the feet, or the palm of the hand, without any rise of outer temperature, there is excretion of sweat, for which physical exertion can not be made responsible. In this tendency to perspiring hands, the slightest psychic excitement, as embarrassment, terror, or anxious expectation suffices to produce profuse hyperhidrosis. In the course of general diseases, such as tuberculosis and in the debilitated condition of convalescence, there often appear during sleep severe perspiration about the trunk and head (night sweats, better sleep sweats), but the skin can also lose its ability to secrete sweat in places, or over the entire body (anhidrosis).

Secretory disturbances of the *digestive glands*, which are to be traced back to nervous influence, are mostly beyond our ken, except that an increased *flow of saliva* may be observed. This almost always appears during nausea. In individuals of a neuropathic constitution, however, profuse salivation appears spontaneously, without any apparent cause, and may last for days, or even weeks.

Many factors make for the view that certain forms of *hypersecretion of the gastric juice and of colitis mucosa* rest upon a "nervous" basis. The first appearance or an aggravation of these phenomena is observed so frequently after psychic excitement, that we can not deny a causal relationship. A proof of the influence of psychic processes on the innervation of the stomach and the intestines is shown also by the appearance, in emotional states, of vomiting and diarrhœa.

Whether, as many suppose, genuine *sympathetic neuralgias* and *visceral neuralgias* actually appear is a very doubtful point. The bodily pains that occur in lead poisoning can not be considered as pure neuralgias, since they are accompanied by spasms of the intestinal musculature.

If in the muscular hollow organs obstructions oppose the peristaltic movements, increased contractions, which can in no way be influenced by our will, attempt to overcome them. This increased exertion on the part of the unstriped musculature is occasionally accompanied by cutting pains. Violent colics, whether proceeding from the intestinal canal, from the uterus, or from the bile ducts or urinary passages may condition an *irritation of the entire autonomous nervous system*, and lead to pallor, salivation, outbreaks of perspiration, dysuria and diarrhœa.

Sharp pains are caused also by disturbances of blood-supply to the intestinal tract, and these may scarcely be brought to consciousness by other paths than those of the abdominal sympathetic.

Of the great list of agents that influence the autonomous nerves and the organs supplied by them (pilocarpine, adrenalin ergotin, caffein, etc., etc.) only two, nicotin, and the secretion of the thyroid glands, will be mentioned here.

On the physiological side it has been pointed out (*Langley*), that nicotin, by intravenous injection as well as by touching the vertebral and prævertebral ganglia, had a paralyzing effect on the sympathetic system of a number of animals—such as the cat and the rabbit—experimented upon. Also the clinical phenomena of acute tobacco poisoning in man (palpitation of the heart, vomiting, diarrhœa, pallor, cold sweats, excessive salivation) show how much the regular innervation in the vegetative paths is influenced by the alkaloid of tobacco.

The troubles lying at the basis of Basedow's disease, such as tachycardia, exophthalmos, the vasomotor disturbances, the tendency to perspire, the strong reaction of the internal organs to psychic processes, must be traced almost without exception to irritation processes in the sympathetic nervous system. It is comprehensible that formerly the morbus Basedowii was spoken of generally as a disease of the sympathetic. Resection of the cervical sympathetic has even been tried as a curative method. If more recent investigations have taught that an increase and abnormal activity of the secretions to which the thyroid glands give rise causes this illness, this corrects the former conception only in so far that there is no primary neurosis of the sympathetic, but that its mode of origin must be sought in an autointoxcatiion, i. e., in the poisonous effects of the secretions of the diseased thyroid glands on the autonomous nervous system.

A detailed account of Basedow's disease, as well as of the vasomotor neuroses (Raynaud's disease, erythromelalgia, angioneurotic edema, etc.) will be given in the next chapter.

X

VASOMOTOR AND TROPHIC DISEASES

BY

HANS CURSCHMANN (Mainz)

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1. VASOMOTOR—TROPHIC NEUROSES

(a) The Vasoconstrictor Neurosis of the Extremities Acroparæsthesia (Schultze)

This disease is the least malignant and by far the most frequent of the vasomotor neuroses; it often appears as a monosymptomatic disease, but far more frequently as a partial phenomenon of the most varied neuroses and psychopathias. It was described first by *Nothnagel*, later by *Bernhardt*, *Frankl-Hochwart* and *E. Schultze*, who gave it the somewhat too one-sided name, acroparæsthesia.

Acroparæsthesia attacks women preferably, but men also none too rarely; among the fifty cases that have come under my observation within recent years, 7 were of male patients. The disease appears between the 20th and 30th years; the majority of my patients were early in the twenties, and then again at the climacteric. But neither childhood nor old age is spared; my youngest case was a girl 10 years old, my oldest a woman of 68.

If we wish to characterize the disease in brief, we must first of all try to settle the difference in the conceptions of *Nothnagel* and *Schultze-Cassier*. While the former in his cases always regarded the *vasoconstrictor disturbance* in the form of pallor, "dying away" often combined with light cyanosis, as the primary phenomenon, and considered the neuralgic-paræsthetic symptoms of secondary importance, *Schultze* and *Cassirer* have, in the majority of cases, found objective vasoconstrictory signs lacking. *Cassirer*, therefore, wishes to draw a rather sharp line between Schultze's form, a primary sensory neurosis, and Nothnagel's form, an objective vasomotor neurosis. I should oppose this division: a very careful anamnesis, and, above all, the *clinical* observation of my cases, have shown me that so far in no single case within my experience have the vasoconstrictor disturbances been lacking though often, naturally, they are present only in slight degree. In consequence of this, I should, with *Nothnagel*, place the *vasomotor* disturbance, as of primary importance, in the foreground of the picture, and define the disease as follows:

Vasoconstrictor neurosis (acroparæsthesia) *is manifested by intermittent spasms in the vessels of the end organs of the body* (*most frequently in the hands and the feet*; *less often in the nose, the ears, etc.*) *which give to these parts a light-bluish, white to wax-white coloring, and which result in the appearance of manifold sensory phenomena.*

Symptomatology.—The time of predilection for the appearance of the attack is the morning hours, usually *before* the patients leave their beds, but often only after the cold bath. Less common is that severe form in which the attacks, like accumulated petit mal attacks, are repeated every hour or more frequently. The attack itself appears rather suddenly; in typical cases, with more or less violent pains, tingling, formication, finally complete numbness and a feeling of being swollen, usually several corresponding fingers of both hands "feel dead" (least often the thumb), less frequently the entire hand; the parts affected often look dead-white in toto, occasionally a little cyanotic. Frequently the pallor is clearly pronounced at the tips of the fingers or the nails, while in the phalanges and the hand, the bluish, often marbled coloration predominates. At the same time, with similar subjective phenomena, the toes and the feet "feel dead," very frequently, also, the legs up to the knees. Besides the subjective symptoms described, there are always present objectively demonstrable sensory disturbances of all qualities, which are not restricted within the bounds of the peripheral nerves, but reach those (first exactly described by *Schlesinger*) of ischæmic sensory disturbance; they are most severe in the distal ends, and decrease rather rapidly in the proximal direction. Besides the sensory, one also finds motor disturbances—usually of slighter degree; either the fingers are stiff, "clammy," "distorted," occasionally to tetanoid stiffness, or the motor disturbance is rather a decrease in dexterity, due to the absence of the feeling of touch and pressure. Often the patients in the attack complain of analogous phenomena, cold, numbness, pain in the *peripheral parts of the face* also, especially in the nose and the ears; then one finds in these parts too the objective signs of angiospasm. The attack itself frequently lasts only a few minutes, on an average, from a quarter to half an hour—in rare cases, which then are accompanied by deep local syncope and asphyxia, for several hours or half a day. After the disappearance of the anæmia there is frequently a reactive hyperæmia, reddening, swelling, and (subjective and objective) heat in the parts affected, which attain high degrees, and occasionally give the semblance of veritable erythromelalgia. This concluding phase of the attack is naturally also accompanied by manifold sensory disturbances.

In the majority of cases, *the exciting causes* of the attack itself can *not* be discovered; it is remarkable, perhaps, that the attacks very often occur in the early morning hours, that is, at the time when physiologically, the action of the heart is weakest, and the blood pressure is lowest. Occasionally, the patients ascribe irritation by cold as the cause. Angiospasms frequently occur in an hysteric attack or as an equivalent of such; it sometimes seems then, as if the somatic impression of the vasomotor attack were the "agent provocateur" of the hysteric manifestation. I have but rarely observed an actually certain psychogenous excitation of isolated vasoconstrictor attacks, whereas they seem to occur far more frequently in angina pectoris vasomotoria. During work or other muscular exertion, the vasoconstrictions—in contrast to intermittent claudication—appear but rarely, they usually even disappear at such times.

This should suffice for the picture of the typical simple cases. A very common abortive type of the disease which *Nothnagel*, with a certain amount of justification, wished to differentiate as a disease sui generis, but which has been neglected altogether, or has not been sufficiently considered by neurologists (*Cassirer*, *Oppenheim*) is the following:

Angina pectoris vasomotoria. This disease appears also in well characterized attacks, especially in the early morning hours. The subjective and objective symptoms of the vascular spasm in the periphery are the same as those described above, only usually rather more severe. There is nearly always this dead feeling in the ends of the extremities, noticed even by the patients; the actual "cadaverous fingers" appear. In the beginning or at the height of the attack, pronounced and mostly *painful cardiac symptoms* are often added: pains and constriction in the heart, marked palpitation of the heart, a feeling of terror, exaggerated to one of utter annihilation, radiating pains in the left arm, in short, *all the symptoms of genuine angina pectoris.* The anamnesis and the objective findings will show us easily that real angina pectoris is not present; usually nervous, hysteric, physically sound, young girls without syphilitic infection are attacked; all signs of aortic or coronary sclerosis are lacking. The disease, accordingly, runs its course without any injury and ends in complete recovery. *Nothnagel* connected the anginous heart troubles with a constriction of the coronary vessels—synchronous with and analogous to that of the peripheral. I have occasionally observed that other vascular areas, too, were affected in such benignant cases (violent unilateral headache, paroxysmal amblyopia in one or both eyes, and finally momentary loss of consciousness). During the attack there is usually no striking change in the heart in such cases; usually tachycardia, very seldom irregularities are present. A disappearance of the pulse in the afferent arteries (for instance in the art. radialis) is never found in benign, non-arteriosclerotic cases. The blood pressure is very evidently, though but moderately, increased in the attack, a result of the increased resistance in the periphery. Ætiologically, I have seen with striking frequency for this form of vasomotor neurosis, psychic traumatisms and the like (for instance, in 3 cases, continued coitus interruptus).

Besides these benign cases, others also occur, in which the angina pectoris *vasomotoria* forms the partial phenomenon or the equivalent of a *genuine coronary-sclerotic angina pectoris* (*Huchard, v. Schrötter, author*). I have observed five such cases, three of which ended fatally, and in post-mortem showed coronary sclerosis. The attacks are quite like those of the benignant angina pectoris vasomotoria. Uni- or bilateral amblyopia appears rather frequently in the attacks (spasm of the art. retinæ). Often, simple syncope and asphyxia of the fingers *without* any angina feeling interrupts the attacks. The systolic (and probably the diastolic also) blood pressure is always considerably increased (about 50 mm. Hg or more in the attack). Usually in these cases, there is no disappearance of the afferent pulse; but unquestionably there are among them forms combined with intermittent claudication, that is arterio-sclerotic dyspraxia. I have mentioned these rare cases at greater length because they urge upon us a careful somatic examination (especially of the heart) in the interest of correct diagnosis in angina pectoris vasomotoria.

Complications of the disease are naturally common and varied. In many, if not in most cases, we must conceive vasoconstrictor neurosis as a partial phenomenon of another neurosis (of general or local character) rather than as an independent disease. Exceedingly frequent is the *syndrome hysteria*, usually of the general, cerebral "feminine" type, running its course with attacks, rarely of the monosymptomatic type. I occasionally observed in such cases that after corresponding treatment, the vasoconstrictory attacks were substituted by genuine hysteric attacks (tachypnœ, singultus, etc.) appearing at precisely the same time. Other neuroses and psychopathias also, genuine neurasthenia, hypochondria, circular depressions (cyclothymia) and catatonia (so-called catatonic attacks, *Hüfler*) may be combined with acroparæsthesias. In somewhat rarer cases, I observed a combination of the disease with the classic cardio-vascular neurosis of adolescence with tachycardia with the "cor juvenum," the drumming heart beats, hard pulse, dermographism, emotional erythema and erythrophobia. These cases, all the symptoms of which point to an abnormal lability of the arterial system, tending towards relaxation, i. e., *dilatation*, are mostly spared by constrictor neurosis. The appearance of acroparæsthesias in scleroderma, erythromelalgia, etc., is common. I have also occasionally seen it in acromegaly. The form seen by *Rosenbach*, which is accompanied by apparent changes in the joints is rare. Sometimes we meet vasoconstrictor neurosis also in genuine tetany, especially in its "formes frustes," the tetanoid conditions; it was observed, too, in hysteric pseudo-tetany by *A. Westphal* and myself.

The **differential diagnosis** is never difficult, if it is possible, as it always is, especially with extended clinical treatment, to establish the presence of the classical vasomotor disturbances, syncope and cyanosis. If we are restricted to the description of the patients or if the symptoms mentioned are but little prominent, various organic and functionally conditioned nervous diseases may evoke "acroparæsthesias" (in the broader sense): polyneuritis, with especial frequency, tabes often enough, multiple sclerosis (in its beginning), etc.; also in the prodromal stage of an apoplectic or embolic attack, we hear the patients complain of recurring paræsthesias of the extremities (which are, however, usually unilateral). The same holds true of all disease conditions that are accompanied by arterio-sclerotic occlusion or a narrowing of the vessels; also in dysbasia arterio-sclerotica, in the prodromal symptoms of diabetic and senile gangrene, we find paræsthetic disturbances of the periphery, which, however, are of less intensity than the *pains*. Finally we must mention the "tetanoid conditions," lightest incomplete attacks of tetany, which subjectively may closely resemble vasoconstrictor neurosis; but in the latter, the classical signs of tetany, *Erb's*, *Chvostek's* and *Trousseau's* phenomena, are always lacking.

All these conditions which are concerned in differential diagnosis, however, in pure cases, are always without paroxysmal vasoconstrictions (excepting cases in which, as in tetany, mixed forms come into question).

Differential diagnosis from Raynaud's disease in its *chronically* beginning form is most difficult: here one is often unable to decide with certainty whether it is a question of a more severe form of benignant neurosis, or of prodromes of malignant symmetrical gangrene. For there are doubtless gradual transitions between these two diseases that are so closely related.

The **pathogenesis** of vaso-constrictor neurosis is not obscure, if one remembers the above description. I consider the attempts to locate the seat of the disease in definite parts of the spinal cord (medulla oblongata, etc.) and, if possible, to assume grosser organic changes, misdirected. The same is true of the hypothesis (*Friedmann* and others), that a neurosis of the peripheral sensory nerves is present. I assume with *Nothnagel* that a vascular spasm in the peripheral arteries is always at the basis of the sensory phenomena; it is certain that the vascular areas are predisposed, physiologically also, to local syncope (for instance in a cold). In acroparæsthesia there develops—mostly upon the foundation of a general neuropathic predisposition—a peculiar lability and irritability of the peripheral vasoconstrictor nerves. It is probable that in this group of "vasomotorics" there exist permanent tonic changes in the peripheral arteries. The acroparæsthetic attacks then signify the paroxysmal increase of this vasoconstrictor disposition. This *vasoconstrictor* disposition, I would regard as a contrasting analogue to the *vasodilator* disposition, briefly characterized above (dermographism, emotional erythema, erythrophobia, etc.). I think this very plausible.

Course and Prognosis.—The course of the simple vasoconstrictor neurosis is usually quite chronic. Attacks and remissions often move in curves, which are at their height in fall and winter, their depth in spring and summer. Such cases are usually recurrent for years and decades. Therefore the prognosis of these cases quoad valitudinem is often doubtful. There may result interference with the vocation of the patient (especially in artists, skilled artisans), but there are also quite sporadic cases, for instance during convalescence from an infectious disease, which after one or more attacks, recover spontaneously and permanently. The prognosis of benignant angina pectoris vasomotoria, especially when it appears as a syndrome or equivalent of hysteric conditions, seems to be comparatively more favorable. Then one often succeeds in setting aside even the vasoconstrictor attacks by simple suggestive treatment. Angina pectoris vasomotoria in genuine angina pectoris must, naturally, be judged differently and far less favorably, especially quoad vitam.

Treatment.—Among all tonic or sedative remedies, arsenic, strychnine, phosphorus, also bromine and digitalis have been suggested. I have seen positive results from a long continued *quinine* treatment (3 times daily, 0.2 chinin. muriat.) and should recommend this most earnestly: besides this —a combination of hydriatic and electric procedures—the use of electric partial baths (as of four cell baths). In individual cases, one can successfully prevent the occurrence of the night and morning attacks by covering the hands warmly at night (as, for instance, in gloves). At the same time, one should never miss the opportunity of exerting a pacifying psychic influence on the nearly always nervous, usually hysterical patients, and of eliminating the hysteria-producing factors that were found in the former life of the patient.

In cases of angina pectoris vasomotoria, which arouse suspicion of a synchronous genuine angina pectoris, the remedies that combat cardiac insufficiency and arterio-sclerosis (digitalis, preparations of iodine as well as of the nitrites) come to the therapeutic foreground.

(b) Raynaud's Disease

Symmetrical Gangrene

This disease, after having been described several times—even by authors of the 18th century—was first comprehensively portrayed by *Raynaud.* It is the classic example of a severe vasomotor and a synchronous trophic disease, and may be characterized briefly as follows: *painful angiospasms appearing paroxysmally, partly with a consequent venous stasis, attacking symmetrically the most distant peripheries of the extremities and of the face, and leading in a shorter or a longer time, to gangrene in these parts.*

The disease is rare, at least in its severely mutilating forms, and attacks mostly youth, even the suckling age (*Cassirer*); women are supposed to be attacked more frequently than men, even though the predominance of the female sex does not appear so decidedly here as in the benignant angioneuroses. In some cases, hereditary and familial appearance have been observed; frequently there is found hereditary neuropathic (also direct vasomotor) predisposition in the previous history of the patients.

Numerous factors have been designated as exciting causes, acute infectious diseases, traumatisms, psychic shocks, cessatio præcox mensium (due apparently to a confusion of the syndrome with the cause), plumbism, morphinism, syphilis (especially hereditary), in short, the most heterogenous conditions that always help out the pathology ætiologically, when the real cause is unknown.

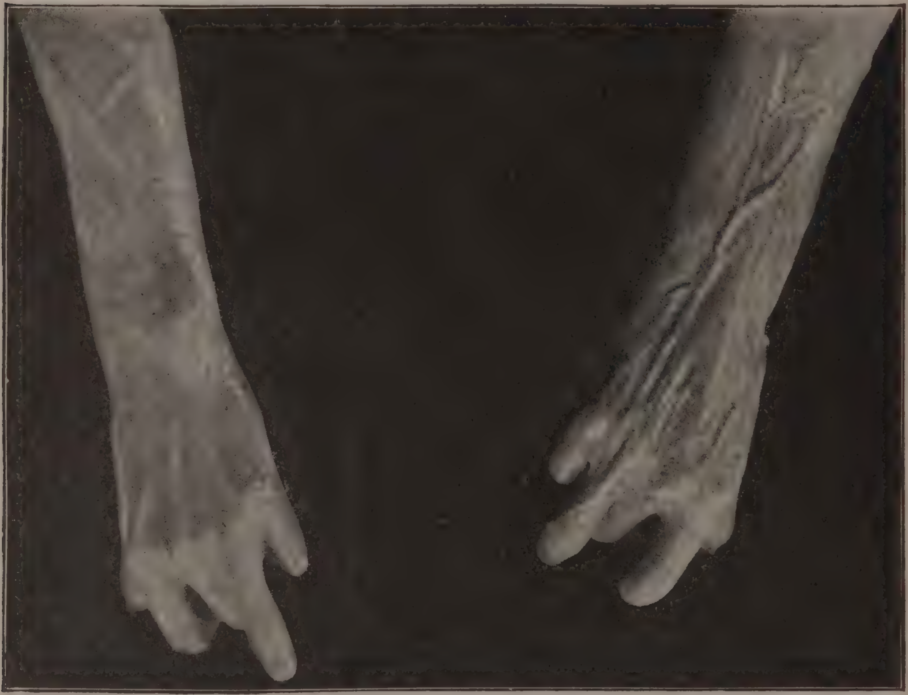


FIG. 192.—Defects in the terminal phalanges in Raynaud's disease. (Medical clinic, Leipsic.)

The typical cases, which lead to gangrene in the distal portions of the extremities, run their course according to *Raynaud's* portrayal (to which have been added the experiences of *Cassirer* and many other authors) in several often sharply differentiated but occasionally confluent phases, the *first* of which is *local syncope*, the *second asphyxia*, and *the last gangrene.*

The first phase, which is frequently preceded by all sorts of light sensory and vasomotor prodromes, often attacks the patient very suddenly; the fingers, especially the distal phalanges, the toes, often also the nose and ear lobe become deadly white, ice-cold, usually anæsthetic, occasionally, too, hyperæsthetic; the nails have a light cyanotic tint; the skin frequently appears a little mottled. Frequently extremely violent almost unbearable pains which may radiate through the entire extremity, characterize the attack in its initial stage, and especially at its acme. Fainting-like or angina pectoris-like feelings are usually absent. There is never fever. The local syncope is said to last usually a short time, a few minutes to hours, and may then be followed directly by the second stage, the local asphyxia; less common is the appearance of a (physiological) exhaustion hyperæmia or a pathological suffusion and swelling after the manner of erythromelalgia.

The asphyxia which takes place in the same areas as the first phase, changes the external picture—usually with increase of pain—as follows: the fingers become cyanotic from the lightest bluish red to a dirty deep slate-blue; they are often spotted; paler in the vola, more deeply cyanotic upon the dorsum; frequently bright red spots are found, for instance on the back of the hand. The parts affected seem bloated, their consistence is increased, they are hard, the skin is tense, often abnormally glossy. This condition may last hours or days. Now in many cases, with increase of the bluish black discoloration the last act follows, *gangrene;* but frequently these purely angiospastic attacks recur for months or years before the gangrene appears.

Gangrene is manifested nearly always by a *dry* shrinking and black discoloration of the parts affected, and ends with a gradual shedding (mutilation) of the same; occasionally it begins with the appearance of large hæmorrhagic blisters, which become transformed into necrotic foci, and finally into total gangrene. As a rule, the mutilation affects only the distal phalanges of the fingers and the toes, more rarely the tip of the nose and the lobes of the ear; the gangrenization of more proximal parts of the extremities, the foot, the leg proper or parts of the trunk (for instance, the skin over the sternum), is quite unusual.

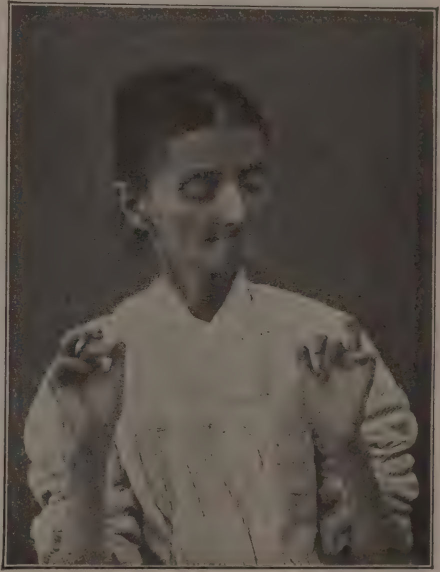


FIG. 220.—Raynaud's disease. (Medical clinic, Leipsic.)

But equally frequent—perhaps still more frequent according to my own experience—are the cases in which the acrocyanotic condition is followed by gangrene of only very small spots in the phalanx that was formerly totally asphyctic; the skin rises in a small blister, little sores develop, which end with superficial indrawn pigmented scars (especially on the finger-tips).

Such small often dot-like scars are found in great numbers on the fingers of such patients; each of these scars signifies, as the patients tell us, the product of an "attack."

While the forms that are accompanied by genuine mutilations often end in recovery after one or a few attacks, in some weeks or months, it seems to me that the forms with lighter attacks and only small gangrenous spots, are essentially more chronic, may persist for years, decades, and the entire life-time.

These more chronic and often also benignant forms of *Raynaud's* disease take us to cases which *Cassirer* would separate as an individual group from typical symmetrical gangrene; to the form designated by him as acrocyanosis chronica anæsthetica, which slowly and gradually leads to permanent asphyxia of the "acra," with anæsthesias, paræsthesias, and all sorts of light trophic disturbances (especially of the hypertrophic kind). I have observed some cases which may be regarded as transitional forms between the above-mentioned chronic, more benignant form of *Raynaud's* disease, and the acrocyanosis chronica of *Cassirer*. In these cases, the acrocyanosis remains permanently, increasing under the influence of cold, in heat more connected with rubor. Pains are rather rare; chronic small whitlows, fissures, occasionally, too, small dot-like necroses are common. Syncope of the fingers appears frequently, especially in the morning; the sensory disturbances are always rather light.

Very frequently these chronic cases run their course with trophic disturbances, which resemble those of scleroderma and arthritis deformans; with gradually appearing atrophies, pointing of the finger ends, the skin of which becomes hard, glossy, and devoid of wrinkles. Bones and joints may—somewhat less frequently—participate in this *gradual* wasting. Frequently there are found, besides, various changes in the nails: they become brittle; there are ridges down and across them; they are subject to pigmentation and manifold deformations.

Among the changes in *nervous* functions should be mentioned the disturbances in sensibility; usually there are (especially in the attack) considerable hypæsthesias and hypalgesias, which naturally do not show the demarcations of neuritic or segmentary, but of ischæmic sensory, disturbances; i. e., they are confined to the asphyctic parts. In rare cases, more complicated, for instance, dissociated sensory disturbances have been observed (also by *Oppenheim*). The sensory disturbances alternate with the fluctuations of the vasomotor ones; this is true especially of the pains which, as we have already mentioned, are never lacking. The higher sense organs may, as will be discussed later, be affected in the attack. Motility always suffers in the attack; the fingers are often stiff, immovable. Reflexes, sphincters, etc., are free from changes. Psychically, we find in patients sometimes—but by no means constantly, as in the benignant vasomotor neuroses—traits of hysteria and neurasthenia, very rarely of Basedow's disease; psychoses have also been observed in *Raynaud's* disease.

Rarer Complications and Symptoms.—Here, we must mention before all the participation of the higher sense organs in the vasoconstrictor attack; sometimes, uni- or bilateral temporary blindness has been observed, which, just as in organic or functional angina pectoris, was caused by temporary contraction of the retinal artery; transitory phenomena of functional loss and irritation on the part of the brain (pareses, spasms) must be traced back to analogous disturbances. Occasionally it has been observed that the afferent artery (for instance the art. radialis) contracted during the attack, that its pulse diminished, that is, became imperceptible; in any case, such a degree of participation by the large arteries is extremely rare.

In some cases, hæmoglobinuria and hæmoglobinæmia have been observed in the attack (*Cassirer*, *Rietschel*).

In very rare cases there have been found, in the hands affected, muscular atrophies, which displayed varying degrees of quantitative and qualitative electric reaction changes.

Of **complications** of symmetrical gangrene, that is, of diseases to which are added Raynaud's symptoms, we must mention first *scleroderma;* especially the forms of this disease that are chronic and are accompanied by severe atrophy of the extremities and the face, frequently exhibit vasomotor disturbances and smaller or larger gangrenization; I have also observed mutilations of the end phalanges. Furthermore, in the literature, Raynaud's phenomena are reported in tabes, syringo-myelia, and other organic nervous diseases that have a tendency to trophic changes. But usually in these cases, it will be found to be a matter not of genuine gangrene, produced by remittent spastic vascular occlusion, but of a neurogenous gangrene. Ergotism, according to *Ehlers*, may also lead to Raynaud's gangrene; considering the well-known influence of ergotin on the vasomotors, we can not be surprised at this. The cases in which chronic nephritis and diabetes lead to symmetrical gangrene must be judged with special care, considering the comparative frequency with which the gangrene of such patients rests upon *arteriosclerotic* processes.

Pathological Anatomy.—The pathogenetically significant findings are scant and very variable. In pure, typical cases, the brain and the spinal cord were probably always found normal. In the peripheral nerves, various changes, genuine neuritis, pure degeneration phenomena, etc., have been established. But the findings are very inconstant, and probably to be interpreted as secondary or coordinated lesions. The vessels are usually free from changes; but the presence of all sorts of processes has been frequently established in them, atheromotosis, endarteritis, and endophlebitis (*Dehio*) of the most peripheral vessels, especially pronounced proliferation of the intima, thromboses, etc. It is a question whether these phenomena also should not be regarded as partial phenomena of the general changes in the tissues. Changes in the heart and the large vessels are rather rarely noted in the literature. The anatomic changes of the skin, and the subcutaneous connective tissue, need no further elucidation here. It has already been mentioned that changes in the bones of the parts affected are nearly always found; besides total necrosis, rarification of the spongiosa, cracking of the nail bed, etc., have been observed (*Blezinger* and others).

The **pathogenesis** of the disease is not altogether cleared up. Simple observation teaches that the symmetrical decay of tissue must be related ætiologically to the insufficient or sometimes to the entirely absent blood supply, that is manifested locally by asphyxia and syncope. I, with *Cassirer*, consider quite improbable a neuritic ætiology in which some authors would believe; also the hypothesis that would implicate changes in the anterior gray matter of the trophic centers of the spinal cord.

If we assume vasoconstriction to be the cause of the total death of the tissues, the question arises: how is it that when the vascular occlusion is not even total, there often appears through a spasm a gangrene, which, however, as numerous experiments show, fails to appear even when an afferent main vessel is completely closed for many hours? In order to answer this question we must assume for the parts to be affected by gangrene a certain predisposition to die, "an opportunity for necrosis," as *Virchow* expressed it (cf. *Cassirer*). Now this opportunity seems to me to lie less in the general condition or the constitution of the patient, i. e., to rest on an eventual anæmia, dyscrasias, etc., but it might be evoked by another factor. As pletysmographic examination of the diseased limbs of those afflicted with Raynaud's disease show, every constant normal reaction of the arteries is absent in them (to temperature, pain, emotions, etc.), even during the period that is altogether free from attacks; this condition allows us to assume a *permanent* change in tonus in the form of a vasoconstriction—since in many of the patients (9 and 12 year old boys) arterio-sclerosis was out of the question. *Now that this permanent contraction of the afferent vessels causes a similarly permanent insufficient nutrition of the peripheral parts, is evident, and in this I would see the cause for "opportunity for necrosis" of such parts, to which they must fall prey if the arterial influx is suddenly—even if not for a long time—interrupted by syncope and asphyxia.*

The real cause of the angiospastic disposition (in the widest sense) is not touched by this. It has not yet been proved exactly how it should be explained. It must be a question, as *Raynaud* and after him *Cassirer* assumed, of an abnormal irritation and irritability of the vasomotor centers and tracts. I can not make up my mind to consider single spinal vasomotor centers affected, since the *vasomotor* symptoms at least, as in benignant constrictor neurosis (acroparæsthesia) show quite *universal* distribution (all "acra" of the head, hands and feet). This general irritability of the *vasomotor* organs seems to be directly caused, above all, by a definite, often congenital disposition, that is anomalies of constitution (cf. the frequent appearance in earliest childhood); certain poisons, however (ergotin), and infections also seem to be able to play this rôle.

The *differential diagnosis* of incipient *Raynaud's disease* must take into account above all, that harmless vasoconstrictor neurosis "dead fingers" (doigts morts), which we meet with so frequently as a partial symptom of a general neurosis (hysteria), in women, as has been noticed, as well as the angiospasms, in vasomotor and organic angina pectoris. In these conditions, however, there never appear real trophic or necrotic phenomena; the pains also are usually less marked than the paræsthesias. Frequently, also, these benignant cases are evidently syndromes or equivalents of hysteric attacks.

The diagnostic differentiation of scleroderma from symmetrical gangrene is occasionally more difficult, since, at times, both may appear closely interlocked. But one must emphasize that the Raynaud symptoms, above all necrosis and gangrene, always appear only in *advanced* cases of scleroderma, which in themselves no longer cause any diagnostic difficulties; slighter vasomotor disturbances are, as a matter of fact, found early—if not very frequently—in this disease. In a differential diagnostic respect, there must be considered, moreover, the cases of spontaneous gangrene in young persons, very rare in Germany, which are supposed to occur as a consequence of intoxications and syphilis (*Oppenheim*).

Important rather than difficult is the differential diagnosis from gangrene and other trophic changes that result from *spinal* diseases, especially from syringomyelia of Morvan's type and hæmatomyelia. The necroses and mutilations that may appear symmetrically in these diseases also, usually run their course without characteristic angiospastic attacks, and very frequently almost *without pain.*

Gangrene with arterio-sclerotic and embolic vascular occlusion (in diabetes, endocarditis) may externally resemble Raynaud's disease; an exact consideration of the fundamental disease on the one hand, and the absence of typical Raynaud's attacks on the other, will usually make a clear diagnostic differentiation possible. Intermittent claudication, that is, dyspraxia angiosclerotica, indeed, exhibits in its violent pains a symptom like that of incipient symmetrical gangrene, but is distinguished from it most clearly by 1. the *permanent* absence of the pulse in the afferent arteries, 2. its appearance *only* after motor activity, and 3. the absence of *externally* visible vasomotor changes in the limb concerned (especially of acrocyanosis).

Leprous mutilations as the partial phenomena of a usually already advanced leprosy, will rarely cause differential diagnostic difficulties. Spontaneous cutaneous gangrene which appears especially in hysterics, will generally be recognized as an "artefact" (self-induced); in the cases known to me, this was easily and surely ascertained. But in case there should be no artificial (self-induced) neurotic forms of gangrene, the differential diagnosis from *Raynaud's disease* may cause difficulties. In rare cases, certain chronic general diseases of the joints, above all, incipient polyarthritis chronica deformans (*Heinr. Curschmann*) is to be considered in differential diagnosis; since the latter disease in its beginning, may occasionally produce only violent pains and trophic disturbances in the nails. The changes in the joints that appear later make the diagnosis sure.

Prognosis and Course.—In many typical cases, the course of the disease is, as has been already described, rather acute: in some weeks or months, after the appearance of the gangrene the disease may come to a permanent standstill. Many other cases, especially those with lighter trophic consequent phenomena, have an essentially more chronic course, and may persist for many years. The disease may for a long time—for months, even years—confine itself to purely vasomotor symptoms, finally to produce gangrenous phenomena.

The prognosis quoad valitudinem, therefore, if often, is not always favorable; on the other hand, the prognosis quoad vitam is always good.

The **treatment** on the one hand, must employ the general tonic measures of climatic and medicinal kinds (ferrum, arsenic, iodine), on the other, must seek to improve the local vasomotor disturbances; the latter purpose is served by warm baths, *careful* massages, and, above all, by mild electric baths (for instance, four cell baths), from which I have seen decidedly good effects. One should try also quinine, which is so very effective in benignant vasoconstrictor neuroses. Whether a careful Bier's hyperæmia, which I have found useful in the above troubles, has a favorable effect in Raynaud's disease must be proved by trial.

(c) Scleroderma (Scleroderma Adultorum)

Like most other angio- and tropho-neuroses, this rare disease, also, appears preferably in women, usually beginning in young adults; but cases in babies at the breast and in the aged have also been described; in children between 5 and 15 years, the disease is, comparatively speaking, not rare.

The acme of the disease is characterized by *atrophy, induration, and anomalies in pigmentation of the skin, which is tightly attached to the underlying tissue, which tissue itself gradually becomes atrophied. In most cases, the disease attacks the face, neck, shoulders, breast, and the forearms rather symmetrically* (symmetrical scleroderma); *far less frequently it spreads diffusely over the entire body* (scleroderma diffusa). *The distribution of the process in blotches or stripes is rather rare* (sclerodermie en plaques, en bandes).

It is customary to assume three stages in the disease: 1. *hard edema,*

2. the *indurative stage*, 3. the stage of actual *atrophy*. Naturally, these stages are not sharply divided during its course, and pass into one another; it is very common to find indurative and atrophic changes appearing *simultaneously* in the patients. Some authors describe another stage which appears before the first, stadium prodromale sive nervosum, in which general nervous, neuralgic and rheumatic troubles are supposed to stand in the foreground; but to this stage we can assign neither constancy nor specific characteristics.

The common form of the disease, the somewhat *symmetrical scleroderma* usually appears, rather slowly, insidiously, and in blotches, in the hands even diffusely. The stage of hard edema may frequently have preceded, as usually the patients reach the physician only after the appearance of the indurative changes. The edematous condition seems frequently to begin acutely: face, breast and arms, less often the trunk and legs, become swollen (without, however, the vicinity of the eye seeming to be attacked with exceptional violence, as it is in nephritis and erysipelas). The skin displays from the first a very hard smooth surface; it is impossible to raise folds upon it; the pressure of the finger makes no dents. The color of the skin need differ but slightly from that of the normal, but it may appear alternately red and mottled; sharp contrasts are the rule. The temperature of the skin is rarely increased, usually somewhat lowered. Through the abnormal tension of the skin, the face becomes rigid, incapable of expressing emotion. This peculiarity, together with the swelling—which does not change the form of the face by distortion, but rather concentrically—the marble hardness, and the snow-white, rose-red coloring may, as I observed, give head and bust the appearance of a plump doll's head. The edematous stage may—post or propter therapiam—retrogress altogether for the time being. Usually, however, the *stadium indurativum* follows directly. In this, the hardness of the skin together with marked changes in the color and pigmentation, is most prominent. Even in the more symmetrical cases, or in those which

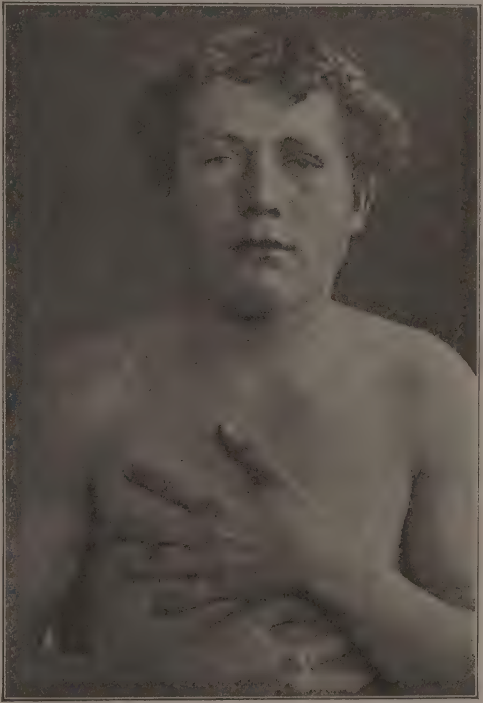


FIG. 221.—Scleroderma in the stage of hard edema. (Leipsic medical clinic.)

later become diffuse, the sclerosis is distributed in spots or stripes; these spots either pass simply over into the normal skin, or are walled off, as it were, from it. "The skin is in some measure protuberant, or is flat, or somewhat depressed, on the surface (usually) smooth, or (less often) covered with wrinkled, thin scaled epidermis, has fatty-like glossiness, or is dull, dead white, like wax or alabaster, or from pink to brown red, sometimes covered with freckle-like white, pigmentless points and dots, and yellow to dark brown (even bronze brown) spots of pigment" (*Kaposi*). Whereas the skin itself does not yet seem thinned, it is already shrunken too tense; it fixes the face, inhibiting movements, the opening of the mouth, the play of the nostrils, the entire expression; it also fixes the hands and the forearms often in a half flexed contracture, occasionally, too, the neck, so that the skin which is shrunken over the platysma draws the head down on the breast. Occasionally deep hard furrows pass through the skin, "as if pulled in by a tight subcutaneous band" (*Kaposi*).

The temperature of the skin is usually somewhat lowered, less often quite normal, corresponding to the subjective feeling of cold that often annoys the patients. The sensory functions, above all, the sense of touch, are usually surprisingly little impaired, often absolutely normal (in contrast to Raynaud's disease). It is not surprising that cold is comparatively little felt by this cool skin. The secretion of sebum and perspiration is supposed to be but little affected during this stage.

Even at this indurative stage of the process, a temporary and partial retrogression is still possible, even though it is rare. This hope naturally disappears with the advance of the *atrophy*.

To this *atrophic* stage belong most cases that come to the hospital and clinic and therefore become more generally known; of the 10 to 12 cases observed by me, fully 8 were in the atrophic phase. But it must again be emphasized (and is already evident from the description) that atrophic processes are always found even in the indurative stage. Now the patients present a highly characteristic picture (cf. Fig. 222): the face in toto is considerably smaller, the nose thin, emaciated in its cartilaginous portion, the mouth shrunken, so that occasionally the teeth are permanently visible, the lips and teeth can be opened only a little, the chin recedes; the lower jaw becomes small and depressed because of the loss of teeth which often takes place early. Everywhere the skin, which is as thin as paper, smooth, hard and glossy, lies fast and immovable upon the bones, displaying the most varied losses of pigment, or accumulations of vitiligo-pallor, ranging even into deep yellow brown: taken all in all, the picture is that of a rather well preserved head of a mummy. Furthermore, the skin of the neck, shoulders, breast, less frequently of the abdomen, participates in the atrophy, everywhere inhibiting the mobility by being shrunken and tense. Often there develops through the shrinking of the skin and the muscles, a passive kyphosis of the cervical and thoracic portion of the spinal column, more rarely scoliosis. Especially pronounced is the wasting of the skin in the fingers, the entire hand becomes as small as the emaciated hand of a child,

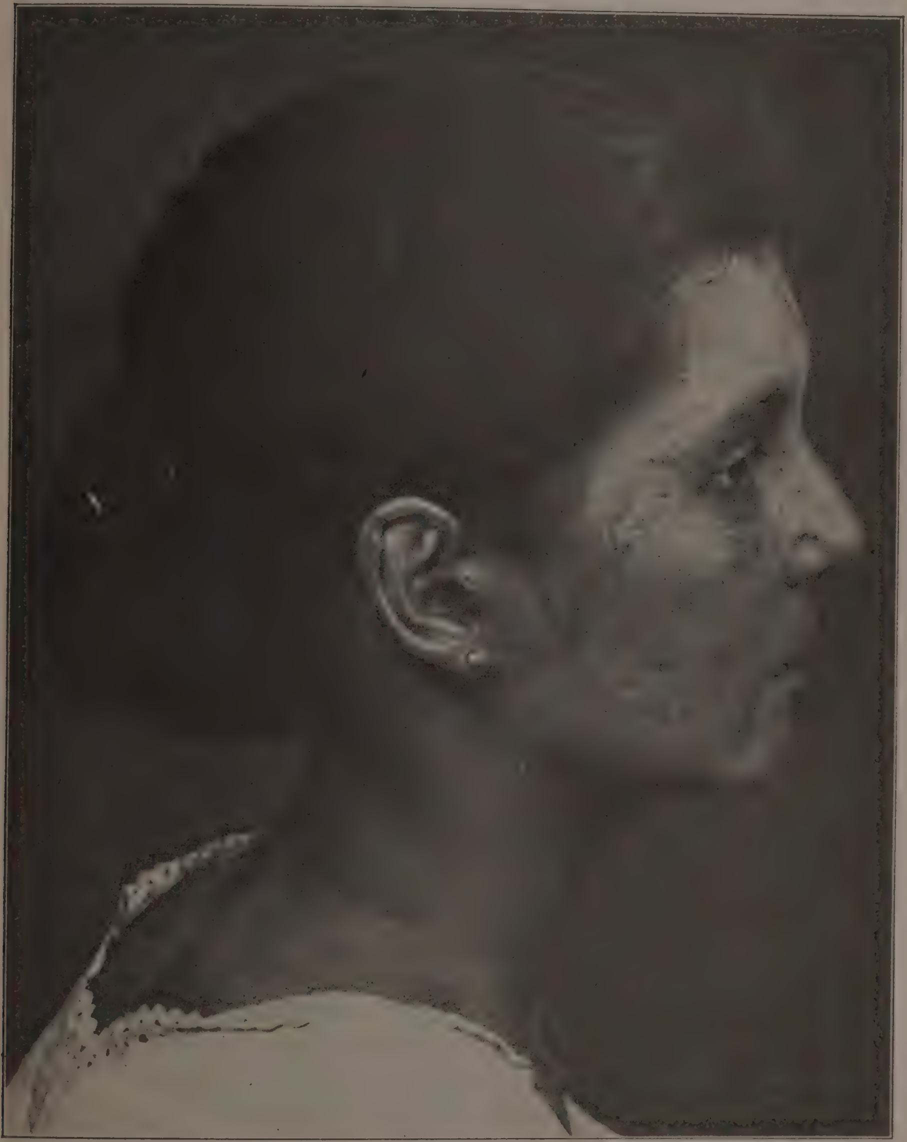


FIG. 222.—Scleroderma with shrinking of the nose, the lips and the chin, pigmentations and vitiligo. (Medical clinic, Leipsic.)

the fingers become pointed, fall into permanent flexed contractures through the shrinking of the skin; thus subluxations and other anomalies of position develop as in the most severe arthritis deformans. (Sclerodactylia, a *symp-*

tom of the disease that has been classified probably unjustly as a special form of the disease). The changes in the skin elsewhere are the same as those in the face, only here the pallor, the cadaverous appearance is more prominent. In the nails, there are always manifold trophic disturbances. Small areas of dry necroses, even painful gangrene in the end phalanges are not rare. Similar disturbances appear, though far more rarely, in the legs and feet.

The atrophy in this stage affects as a rule also the fatty and other subcutaneous cellular tissues, the ligaments and bones; the bony change has often been proven in recent years by means of the X-rays. The wasting of ligaments, joints and bones, produces ankylotic contractures; I have also observed sclerodermic stiffness of the vertebræ. Genuine muscular atrophies, i. e., those accompanied by severe disturbance of motility are strikingly rare, though they have been observed in some cases (*C. Westphal*). Even in the "homme momie" of *Grasset*, in spite of the high degree of muscular attenuation, I convinced myself of the comparatively good gross strength, that of the muscles of the arm, for instance. The motility disturbance in sclerodermics is usually only a consequence of the shrinking and fixation of the organs of movement.

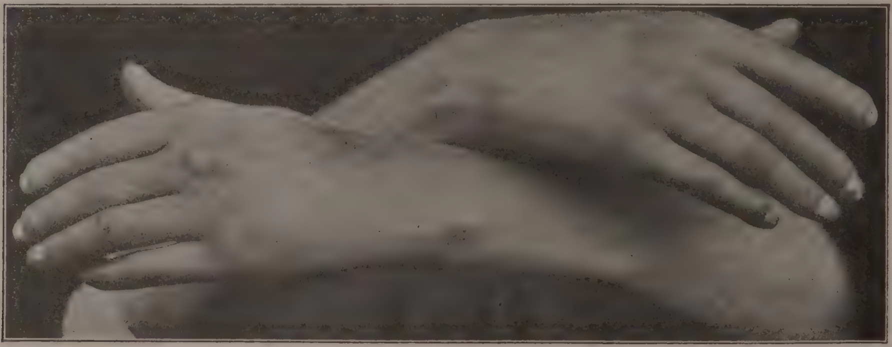


FIG. 223.—Scleroderma with sclerodactylia of the right hand. (Medical clinic, Leipsic.)

The case described by *Grasset*, a peculiar brown dried-up dwarf, is the prototype of general *diffuse scleroderma*, probably the rarest form of the disease. All the changes in skin, bones, and joints, that have been described, attacked, in him, the entire body; the pigmentations were especially marked. These pathetic creatures are really like living mummies.

Even in such cases, the sensory disturbances are always strikingly slight or altogether absent, except that pains are frequent, especially when the joints are affected and upon the appearance of necrosis. There is always present the feeling of tension and restriction on account of the shrinking of the skin. Phenomena of functional loss in the cerebral nerves and the rest of the nervous system are scarcely ever found in any forms; the sphincters function normally;

the sexual function (especially menstruation) is usually lost. The organs of circulation rarely exhibit disturbances; arterio-sclerosis is rare. Once I found in the heart of an older patient persistent ventricular bigeminia with apparent bradycardia of the pulse, phenomena which could scarcely be traced back to specifically sclerotic changes in the heart; the same is probably true of the other cases of disturbances of the heart in scleroderma, described (rarely) in the literature. On the other hand, vasomotor disturbances, syncope, and asphyxia occur rather frequently in all stages of scleroderma; also in genuine Raynaud's disease; the combination with symmetrical gangrene has been already described in this chapter. Also in respect to the functional (plethysmographically determinable) condition of the vascular reactions I found areflexia similar to that in *Raynaud's disease.*

Macular (circumscribed) *scleroderma* is a rarer and more harmless form of the disease. In various places on the skin of the body, rarely in one place alone, there appear round macules from the size of a finger-nail to that of the palm of the hand, or stripes and bands which stand out a little at first and later sink in. The periphery of these macules shows all sorts of color tones: yellow, brown, bluish, gray, frequently lilac (so-called lilac ring). Towards the center of the macule, the color disappears until the spot is pure white, and, at the same time, harder, sclerotic; here the skin is tightly attached to the parts beneath and can not be lifted. Because of its peculiarly transparent appearance, it has been compared to bacon rind (*Lesser*). Gradually in this form, atrophy of the skin develops, which then becomes as thin as paper, smooth, glossy, and unwrinkled. The hair falls out, the secretion of the cutaneous glands is supposed to cease. In two cases that I observed, the atrophic stage appeared very quickly, in a few weeks, so that, from the description of the patient, I could almost doubt whether a clearly sclerotic stage had preceded.

Rare Cases and Complications.—In rare cases, a synchronous atrophy of the mucosa and the submucosa of the tongue, gums, pharynx, and larynx, with stenosis of the same, has been observed (*Arning*). Sometimes it has been observed that the scleroderma was closely confined to the boundary of a cutaneous nerve or segment; *Krieger* and I saw in a spondylitic a broad sclerodermic stripe at the upper boundary of the sensory paralysis.

In one case (*Westphal*) sclerotic foci were found in the brain also.

Occasionally one finds cases in which the changes in the joints, ankyloses, etc., appear strikingly early, seem virtually to rush ahead of the skin sclerema; here, under certain conditions, the differential diagnosis from arthritis deformans with atrophy of the skin, is very difficult. Scleroderma may also be combined with ankylosed vertebral stiffness of Bechterew's type. Of complications of scleroderma, one must mention before all Basedow's disease, occasionally found together with tetany, manifold forms of vasomotor neurosis (ranging from benignant acroparæsthesia to Raynaud's gangrene), hemiatrophia faciei and supposedly also Addison's disease (probably confusion with the severe pigmentation of the diffuse cases); also neuroses, hysteria, cyclic depression, neurasthenia, and other affections have been frequently observed as concomitant diseases.

Anatomy.—The central nervous system, brain and spinal cord, were usually found normal, the changes established in isolated cases (sclerotic foci in the brain, small cavities in the gray axis of the spinal cord, etc.) are certainly not concerned *ætiologically*. The peripheral nerves also were usually intact; the changes occasionally found, simple degeneration, parenchymatous neuritis, perineuritis, should rather be regarded as coordinated or secondary phenomena. The sympathetic system is also found intact. The vascular system, especially the arteries, were mostly altogether normal; especially was it free from atheroma; but exceptions to this occur.

The histological changes of the skin are very differently described; usually there was found a coarsening and thickening of the reticulum of the connective tissue of the skin (K*aposi*) "so that the homogeneously constituted, coarse-fibered and narrow-meshed tissue of the cutis reaches close up to the fascia and periosteum, and clings to these without any loose intermediate layer." The elastic fibres have been described by many investigators as considerably increased, by others again as normal or diminished. In the corium, increase in the pigment was invariably observed. The lymphatic ducts and also the sweat glands were found constricted, pressed together; there was always great disappearance of the cutaneous sebaceous tissues. A hypertrophy of the smooth musculature of the skin was found as a rule. The cutaneous nerves were usually normal. Just as regularly present were changes in the cutaneous vessels; on one hand, simple contraction due to the presence of the sclerotic connective tissue and the layers of the lymph cells, on the other hand, diseases of the vessels themselves, which affected all of their three layers (peri-, mes-, and end-arteritis, Hoffa); veins and capillaries were also found changed.

In the muscles, bones, ligaments, etc., participating in the atrophy, corresponding changes of partly simple atrophic, partly secondary inflammatory nature were found.

Among the internal organs, the thyroid gland is of peculiar anatomic interest; there have been found (cf. *Cassirer*) very different processes; increase of the connective tissue, cystic dilatation, occasionally general atrophy (Hektoen), all in all, nothing pathognomonically certain.

The **pathogenesis** of scleroderma is as much discussed as it is obscure, like that of the other trophic neuroses. The vascular disease, the peri-, mes-, and end-arteritis, which, however, as we have seen, is not constant, has been assumed to be pathogenetically of primary importance. Other authors (*Wolters*) see in the disease, in contrast to *Kaposi*, a genuine chronic inflammation; others again, accuse the chronic lymphatic disturbance of being the ætiological factor.

Coarse organic lesions of the central and peripheral nervous system have also been brought in ætiologically (*Bruns* and others); the observation that sclerodermic changes in the skin in case of affection of the separate roots and peripheral nerves have been found in the distribution area, has given cause for this. I do not believe that this is correct. For this very rare *symptomatic* scleroderma is essentially different from primary scleroderma. An affection of the sympathetic nerve has also been assumed as the cause of scleroderma (*Brissand*). But one may be quite sure that *Cassirer* is right, when he assumes that scleroderma is not evoked by coarse changes in the nervous system.

The thyroid gland theory, the assumption of a dysthyreoidism, which is based upon the syndrome scleroderma and Basedow's disease, is improbable and not satisfactory, precisely because the simultaneous occurrence of the two diseases or even of single symptoms is comparatively rare. Of the 12 cases known to me, only one was combined with Basedow's disease. To conceive of scleroderma as a disease evoked by concomitant disturbances in the adrenals, is not possible, in spite of the frequent, occasionally diffuse, bronze pigmentation, as the other symptoms of Addison's disease, especially the *sudden* cachexia and hypotension of the blood pressure, are always lacking. *Strümpell's* hypothesis of a possible pathogenetic rôle of the hypophysis (on the basis of a certain, but only superficial, antagonism to acromegaly) has not been proved either.

Nothing remains for us but to assume with *Lewin* and *Heller*, *Cassirer* and others in scleroderma a possibly organic, more probably only functional disease of the vasomotor and trophic tracts and centers, the true nature of which is as yet obscure, that is, to interpret scleroderma as a tropho-neurosis.

The **diagnosis** in developed forms is always easy, since the picture of typical, symmetrical or diffuse scleroderma will impress indelibly any one who has once seen it. Differential diagnosis in the beginning of the disease, especially in the stage of edema, may be more difficult. Here there may be confusion with inflammatory changes, dermatomyositis, chronic erysipelas, or with chronic edematous changes (myxedema). The striking hardness and anomalies in pigmentation, however, will soon bring about a recognition of scleroderma. If the vasomotor disturbances, as occasionally happens, are very pronounced, the differentiation from Raynaud's disease may cause difficulties, especially since, as we have seen, a combination of the two diseases is not of infrequent occurrence. The spread of scleroderma *on the surface*, however (for instance in the face or the breast), is a pathognomonic symptom which will usually make diagnosis possible.

Addison's disease, of which one may think when there is diffuse pigmentation, always lacks the hard, atrophic condition of the skin; its malignant symptoms, moreover, are foreign to scleroderma (gastric and intestinal achylia, lowering of the blood pressure, *sudden* cachexia).

Differential diagnosis relative to chronic arthritis deformans and its various forms is important. It almost synchronously with the articular changes exhibits cutaneous and muscular atrophy, later atrophy of the nails, bones, etc. It is important that the sclerodermic *hardness* of the skin does not occur in arthritis deformans typica, nor the changes in the skin of the face, but there are, as I have observed, cases of sclerodactylia in which multiple changes in the joints and the skin appear almost simultaneously.

Macular scleroderma may, under certain conditions, be confused with keloids, certain leprous-like changes in the skin (morphea atrophica), changes in the skin over the primary atrophy of the bones, etc. (*Sudek*). In all these diseases, however, the hardness of the skin is lacking. The same is true also of xeroderma pigmentosum, which is concerned in differential diagnosis, but exhibits also teleangiectasias, cutaneous tumors, etc.

Course and Prognosis.—The course is always chronic. The disease extends over years and decades. The atrophic stage is always incurable; but recovery in the indurative stage has been observed. In contrast to the other tropho- and vasoneuroses, scleroderma usually leads to a very gradually developing *cachexia*, which may be favored by impairment in chewing, respiration, etc. This cachexia may do much to further death on account of an intercurrent disease. Therefore the prognosis of the disease is nearly always unfavorable quoad valitudinem, but must be made cautiously also quoad vitam. Naturally the prognosis of "sclerodermie en plaques" is to be judged far more favorable.

Various attempts at **treatment** have been made. Strengthening and tonic remedies (iron, arsenic, iodine, diet, rest) are naturally always recommended. Good results have been shown from warm baths, steam baths, etc. On the ground of my own experience, I should earnestly recommend before all *thiosinamin* or *fibrolysin* (*Mendel*), used regularly and for a long time, and *careful massage;* thus it is certainly possible to make the sclerotic skin more pliable and to loosen contractures. It seems to me doubtful, judging by my own experience, whether the constant current is effective; perhaps one ought to try *Bier's* hyperæmia.

(d) Neurotic Edema

(*Edema circumscriptum acutum* (*Quincke*) *and other forms*)

Under the concept of *neurotic edema*, we wish to collect those cases of acute or (less common) chronic edema, for which 1. there is no general or local organic cause (cardiac and kidney troubles, anæmias, cachexias, on the one hand; vascular occlusion, local inflammation of all sorts on the other);

in which we 2. may exclude *organic* diseases of the nervous system (cerebral and spinal paralyses, above all, syringomyelia, tabes, etc.); and which 3. by their frequently fitful appearance, quick disappearance and finally also by their combination with psychic nervous phenomena, remind us of the symptoms that we find in all parts and in all functions of the body and—often judging only per exclusionem—are usually diagnosticated as "functional" and nervous (in the broadest sense).

These edemas are of quite manifold kinds. It is not sufficient to group them all under one, and that a rather rare, single form, Quincke's edema, as is most frequently done. The most common form of neurotic edema is *evanescent, nervous edema* (*especially of climacterics*).

The disease is nearly always the partial phenomenon of a general neurosis of hysteric, neurasthenic, or hypochondriac, above all, of vasomotor type, as we find it in women in the climacteric state (especially severe in artificial climacterium præcox). Quite like the general nervous phenomena, the edema is by no means confined to the few years of the "change," but usually lasts many years, sometimes a decade after the cessatio mensium. Furthermore, these edemas may appear even in the second to the fourth decade, just as hysteric and vasomotor disturbances of other kinds occasionally appear years before the climacteric and only become especially pronounced at its beginning, and then appear with especial frequency *at the time of menstruation.* This *menstrual* nervous edema is, however, considerably rarer than the climacteric. The disease is characterized by the appearance of quite temporary, *not circumscribed* but *diffuse swellings which pass gradually and imperceptibly over into the normal skin*, and are usually rather pale, occasionally, too, of normal color, but not bluish or suffused, and cause no subjective phenomena besides a slightly bloated feeling, and paræsthesias. Occasionally they are combined or alternate with vasoconstrictor acroparæsthesias. Their favorite seats are the *hands* and the *forearms.* They usually appear in the morning hours, and disappear after a short time (about one-half to several hours). These edemas are often so *slight* that they would be overlooked by the physician, if they were not, at least by sensitive patients, always felt and complained of as something alarming. They are very stubborn, and recur persistently, just as the other forms of neurotic edema. They need no special treatment, since in the cases concerned, the nervous phenomena of other kinds usually stand indisputably in the foreground.

According to what has been said above, the *diagnosis* is easy. In spite of its close connection with the other forms of nervous edema, especially with Quincke's circumscribed edema, we must separate it from the latter, since it usually lacks the main characteristic of the latter, the appearance of *circumscribed* patches and planes, and frequently lends only a slightly *bloated appearance* to the parts affected. But, as a physician, one must *know* this form in order to be able to destroy with a good conscience, the differential diagnostic fears of the patient concerning organic edemas.

Edema circumscriptum cutis (*Quincke*, 1882) exhibits the following phenomena: in circumscribed places in the skin and subcutaneous cellular tissue, there appear usually suddenly (within a few minutes) less often gradually, swellings of a typically edematous nature; the pressure of the finger always leaves a depression. These circumscribed edematous spots vary in size from that of a quarter of a dollar to that of the surface of part of an extremity (for instance, the forearm); they are usually somewhat reddened, especially at the periphery, while the center is often paler; less frequently the entire edematous part seems pale, or even normally colored; in some cases, small sugillations were observed.

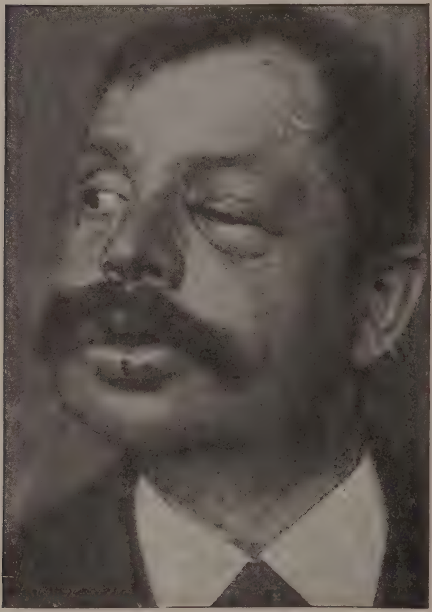


FIG. 224.—Œdema cutis circumscriptum. (*From Moritz.*)

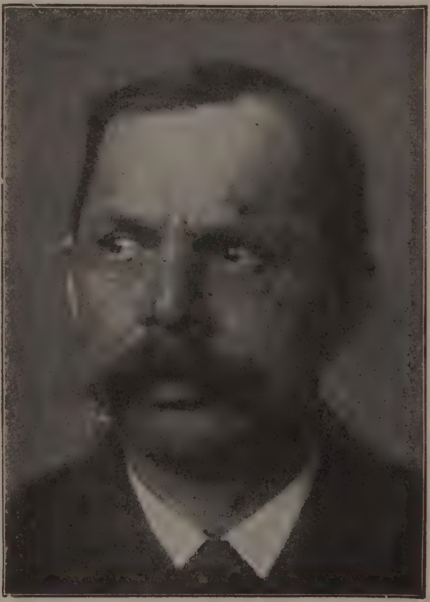


FIG. 225.—The same person as in Fig. 261 after the attack passed. (*From Moritz.*)

Sometimes the disease appears restricted to one isolated spot; but just as frequently, there is a simultaneous multilocular springing up of patches and planes, occasionally, symmetrically as in erythema exudativum. The edema is localized preferaby upon the face (especially the *eyelid*) and the extremities, less frequently upon the trunk. But occasionally also, the mucous membrane of the mouth and throat, the larynx, the conjunctiva, even the mucous membrane of the stomach and the intestines are involved. The subjective phenomena consist only in a bloated feeling, less often, in heat, itching, etc. Though the general health suffers but little, the mood of the patient is affected, as is natural enough, in the face of this inexplicable disease. There is scarcely ever fever. The edematous spots usually pass away as quickly as they come, that is, in minutes to hours, at most days, after their appearance, and apparently never leave traces.

The disease affects preferably young adults, and the male sex. It is certain that in many cases a *specific* hereditary predisposition is present, just as in other vasomotor neuroses—in others (the majority) only a general neuropathic disposition. We know little of the *ætiological* factors. Rheumatic infections, exogenous and endogenous intoxications, light organic and psychic traumatisms, have all been alleged, though no one of these ætiological factors was proved to be constant. I should agree with *Cassirer*, that the predilection cause of chronically recurring urticaria (which is certainly related to Quincke's edema), the effect of certain food stuffs (crabs, strawberries) is not concerned ætiologically in circumscribed edema. Usually no intelligible cause of the disease can be discovered; only it seems certain that the attacks are evoked by the influence of cold.

It is self-evident that we will not identify the edemas that occur so frequently in syringomyelia (especially in the stadium incipiens), tabes, and other organic diseases of the spinal cord and the brain, with Quincke's edema; nor must the edemas in certain neuroses, for instance the frequent edema of the eyelids in migraine, temporary edema in Basedow's disease and myxedema, as well as in various psychoses, be confused with Quincke's disease picture, but should, as is done by *Cassirer*, be designated as "symptomatic nervous edema."

Into this category come also the cases of temporary or (more frequently) long-lasting edema in other vasomotor and trophic neuroses. This has already been said of vasoconstrictor neurosis (acroparæsthesia). But sometimes edema also appears in those places upon the body attacked by Raynaud's disease, erythromelalgia, and especially scleroderma; in the latter case the condition has been called "scleredema."

Paroxysmal disturbances in secretion must be mentioned as rare symptoms of Quincke's edema. Furthermore, one has observed as an equivalent or a syndrome, attacks of strikingly transient vomiting or profuse diarrhœas or a suddenly appearing cold with very watery excretion; polyuria, oliguria, or paroxysmal hemoglobinuria have been described. Meningismus appearing in frequently recurring attacks has even been interpreted as an equivalent of Quincke's edema (*Ullmann*). It has seemed plausible to assume this analogy for many cases of migraine, Ménière's vertigo, and even epilepsy. But it seems to me that these attempts at explanation go too far and are deficient in exact proof.

It is certain that Quincke's edema is closely related to *chronically recurring* (more or less febrile) *urticaria*, especially to urticaria gigantea. Doubtless there even exist very gradual transitions between the two diseases. Thus I observed a 36 year old man, not nervous, who had first suffered from typical recurring urticaria, appearing as small reddened severely itching wheals, about as large as a quarter, which after some weeks increased in size until the typical, scarcely reddened and slightly itching, circumscribed edema of the entire forearm, the lips, or one-half of the face, had developed. Such individuals always show vasomotor over-excitability, especially urticaria factitia, and usually respond to every small blow with a local wheal.

Pseudo-periostitis must furthermore be noticed as a rare localization of *Quincke's* edema, which had already been described by *Quincke* and has recently been described by *Herz;* there suddenly appears over long or flat bones, especially over the sternum, a rather indolent swelling, which disappears quickly and then recurs.

Somewhat more common and diagnostically very important, is *intermittent hydrops of the joints*, our knowledge of which we owe to *Moore*, and, more recently, to *Schlesinger*. Of the two forms of hydrops articuli distinguished by them (the *symptomatic and the idiopathic*) only the latter may be designated as a certainly non-organic nervous syndrome. The affection which afflicts men and women equally, preferring nervous young persons, is manifested by sudden rapid swellings either of one or (more rarely) of several joints, which are mostly painless, combined only with a feeling of tension, and quickly disappear after one or more days. The knee joint seems to be attacked with peculiar frequency; there is no fever in the pure cases. The skin over the joint is usually not reddened; but occasionally there is found acute edema also of the part of the cutaneous region concerned. In many cases, articular hydrops recurs with a certain degree of regularity at definite times, for instance in one of *Reisinger's* cases, every 13th day, in other cases, quite irregularly. Ætiological factors or excitant causes can not be established; slight automatisms may be concerned. Occasionally the attacks were observed in combination with other nervous disturbances of the vasomotors and of secretion; with polyuria, general hyperhidrosis, asthma nervosum, recurring iritis (*Oppenheim*), and others. It must be stated that intermittent articular hydrops is combined with other neuroses, for instance with hysteria and Basedow's disease.

The **differential diagnosis** must consider above all chronic recurring articular rheumatism, which may also come to a climax cyclically (for instance during the menses), but always causes slight increase in temperature. Further must be mentioned the cases of traumatic, often recurring articular hydrops (especially of the knee), and spinal troubles that are accompanied by indolent arthropathias (tabes, syringomyelia).

In the same category as articular hydrops may be included *intermittent swelling of tendon sheaths*, first described by *H. Schlesinger*, which has a course quite analogous to that of the disease first mentioned.

An apparently *primary edema* occurs rather infrequently among *hysterics*. It may occur as edema bleu or edema blanc (*Sydenham*, *Charcot*) (though naturally this schematic distinction can not always be maintained), appears in acute form and with a chronic course, and is often combined with total anæsthesia of the edematous parts. I observed in one case that it could be evoked or at least increased artificially (for instance, by strangulation of the arm). For the rest see the chapter on hysteria.

Still rarer than the acute forms is the *chronic form of neurotic edema*, which may appear quite spontaneously or also after relatively light traumatisms in nervous individuals, and prefers the lower parts of the extremities (forearm, leg proper). The edema may suffer fluctuations and exacerbations, or else remain altogether stable. I observed one case in which after the influence of cold (washerwoman) the edema of both lower arms appeared quickly, and now in its 4th year, it is still persisting in stable form; its color is mostly pale; its consistence very hard. The edema seems often to be combined with pains, paræsthesias, etc. Familial appearance has been observed here also.

Differential diagnostically, there must be considered before all, the sequelæ of chronic thrombo-phlebitis, chronic inflammatory affections of the skin (eczema, erysipelas, and others), organic nervous diseases (neuritis, tabes, syringomyelia, etc.) and finally elephantiasis. The prognosis of this form quoad valitudinem is especially unfavorable.

The *pathogenesis* of all these forms of nervous edema is very obscure. It is doubtful whether the first cause is a venous spasm (and the accompanying congestion). The disturbance in the venous outflow is probably more secondary. If we consider that in experimental animals certain enterotoxins (for instance those from crab meat) produce an increased lymphatic secretion (*Heidenhain*) and furthermore, that the same food-stuffs evoke reflexly in nervously disposed persons chronic recurring urticaria, which is so extraordinarily like Quincke's edema, it is natural to suppose that a primary secretion disturbance, that is, an increase of secretion, is *one* pathogenetic component. The other should be sought in an abnormal permeability of the blood vessels, appearing in attacks and in itself as yet inexplicable, like that which we now have to assume in certain forms of acute (infectious or toxic) nephritis.

The **treatment** is as dark a chapter of the disease as the pathogenesis. Clinicians have recommended dietetic measures, regulation of the bowels, avoidance of certain foods that we have already mentioned, in addition to massage, steaming and faradic baths. They have likewise made various experiments with drugs, using with some success atropine, arsenic, quinine, and strychnine, and many other remedies. On the whole there is not much success in obstinate cases; the light cases which infrequently recur recover, not propter but post hoc, of themselves.

Accordingly, the *prognosis*, especially for hydrops intermittens articulorum and the chronic forms of edema, is rather bad—quoad valitudinem, quoad vitam (except in the extremely rare cases with edema of the glottis) always good.

(e) Erythromelalgia

This disease, the rarest of the typical vaso-motor neuroses, first described by *Weir Mitchell*, attacks almost only adults, and men and women with apparent impartiality (in contradistinction to the other angioneuroses). As in these, the ætiology is absolutely dark, occasionally occupational injuries, acute traumatisms, contraction of cold and drenchings, especially *freezing* and rheumatic infections have been accused. Many of the patients are neuropathics and predisposed persons, as were even the few cases within my observation, of which, for instance, one woman displayed psychic depressions, another severe hysteria, a relatively frequent concomitant phenomenon of erythromelalgia.

Clinical Concept.—*Erythromelalgia is characterized by acute attacks of violent pains, with a consequent circumscribed reddening and swelling of the distal portion of the extremities which follows after a shorter or a longer interval; these appear either strictly in attacks* (*rarely*) *or* (*far more frequently*) *leaving residues and exacerbating, and finally continuing as a permanent chronic condition.*

Symptomatology.—In contrast to the other angioneuroses, the disease preferably attacks the lower extremities, the toes, the ball of the foot, the heel, occasionally the leg proper; in one of my cases, the erythromelalgia persisted above the ankles. The fingers (end phalanges) and hands are attacked somewhat more rarely. Quite acutely there occurs (occasionally with slight increase in temperature and feeling of cold) a swelling of the parts concerned, at first bright red, later darker, which is usually sharply defined from the normal skin. Later the discoloration becomes livid, the temperature lower, the arteries in the suffused area beat strongly, the veins seem dilated. There is violent pain especially prodromally and in the initial stage, but it may persist. Upon testing the sensibility, hyperalgesia always appears. Frequently the spontaneous pain that appears in attacks precedes by weeks the appearance of the reddening and the swelling. The pain is increased by heat, by letting the affected extremities hang, and especially by movement, also by standing and walking; therefore, one of my cases, a servant, has been incapable of work for 2 years, because of the continuous exacerbation of the disease in her vocation. Occasionally the formation of nodules in the area of the erythromelalgia has been observed (confusion with erythema multiforma exsudativum?). The temperature of the skin in the affected areas is always increased. Excessive local secretion of sweat is frequent. In many cases, the affection attacks first one foot and then the other, and later the hands. Occasionally erythromelalgia, like many forms of acroparæsthesia, runs a course of typical circumscribed short attacks, which die out after one or several days or weeks without any residue. This harmless form is supposed to occur especially in hysterics. Far more common, however, are the cases in which the disease becomes either stationary or progressively *chronic*, always, however, being prone to acute exacerbations; the duration of such cases may amount to months or even years, even when the affection, as in one of my cases, was restricted to one part of the skin.

Beside the local symptoms, there are found occasionally, as also in the other vasomotor neuroses, other vasomotor and cardiac symptoms in the attack, for instance, palpitation of the heart, anginous oppression, tachycardia, syncope, headache occasionally in the form of typical hemicrania and others.

The question whether trophic disturbances belong to the picture of erythromelalgia seems answered by *Cassirer*, inasmuch as such disturbances occur, though rarely, as residues and even as preceding syndromes, in the skin (like the "glossy skin" of certain nervous lesions) as well as in the nails and even in the bones. It seems also that gangrene is combined with it in diagnostically certain cases (very rarely and involving but a small area). This fact leads us to the comparatively frequent *mixed forms of erythromelalgia and other related neuroses:* occasionally there are found in cases of vaso-constrictor neurosis, erythromelalgetic blotches, especially at the end of the angiospastic attack; in one case observed by me (severe organic angina pectoris vasomotoria) these appeared after such an attack in a large area on the back of the hand and the forearm, and persisted to the death which occurred 8 days later. Even in the benignant cases (especially in the winter) I have observed violent painful reddening and swelling of the fingers usually attacked by the angiospasm. It seems that Raynaud's disease may also combine with erythromelalgia. It has been asserted that dyskinesia angiosclerotica (intermittent claudication) may be accompanied by similar changes in the skin.

It is supposed that *erythromelalgia* frequently occurs as a *symptom of organic, cerebral, spinal, or even peripheral neuritic affections.* Thus it was found in the paralyzed parts in cerebral hemiplegias, in progressive paralysis of the insane, in sclerosis multiplex, in tabes, in spinal muscular atrophies, and in various forms of injuries to the peripheral nerves (polyneuritis, injuries, etc.). Therefore *Cassirer* is justified in dividing the erythromelalgias into 2 groups: 1. that in which the disease appears rather *symptomatically* as the expression of conditions of irritation in the peripheral nerves (especially of their vaso-dilatator and secretory fibres); and 2. that in which erythromelalgia appears as an *independent* disease of central nervous (spinal or bulbar) genesis; in the latter cases, trophic disturbances occur with comparative frequency.

The *pathogenesis* of the disease is obscure. No gross changes in the central nervous system can be demonstrated in the *idiopathic* cases; it seems also of little avail to invoke the symptomatic erythromelalgia of spinal and neuritic diseases to explain the pathogenesis, since, with the rare occurrence of erythromelalgia in these affections, we can scarcely decide which anatomic or functional disturbance provokes precisely the vasomotor symptom. In any case, we must assume with *Cassirer*, conditions of irritation in certain sensory vasomotor and secretory tracts or centers. Whether it is especially a question of a spinal or a bulbar irritation, as *Cassirer* supposes, has not yet been established.

The **differential diagnosis** is often difficult. Above all, in the beginning, it is frequently difficult to exclude local inflammatory infections (erysipelas, arthritis urica, phlegmons, pes planus inflammatus, etc.). It seems especially hard to distinguish erythema exsudativum multiforme in its not very rare chronic and chronically exacerbating form from erythromelalgia. The very rare erythrodermia (Pick) is supposedly differentiated from it by different localization and the absence of pain. I do not believe that intermittent claudication could cause serious difficulties in differential diagnosis from erythromelalgia. As a rule, only the pains are common to both diseases, whereas the color of the skin in intermittent dysbasia nearly always shows in the attack the signs of anæmia.

Very difficult, indeed, often impossible, is the differential diagnosis from other vasomotor and trophic neuroses, Raynaud's disease, vasoconstrictor acroparæsthesia and neurotic edema, especially of Quincke's form in many cases. There are, as between the rest of these neuroses, also between erythromelalgia and the conditions mentioned, very gradual transitions, in my experience, most frequently with acroparæsthesia, so that the erythromelalgetic condition appears rather as a symptom of the other neurosis, than as an independent disease. Finally, we must consider an affection of great importance in differential diagnosis from erythromelalgia—the sequela of local freezing and frost bites (chilblains), which even have some peculiarities in common with erythromelalgia (exacerbation in heat, when the limbs are let hang, etc.).

Treatment has been attended by no noteworthy results. Of internal remedies, among the sedatives, morphine has been praised above all others; furthermore, the usual antirheumatics and tonics (iron, arsenic). Among external measures, cold compresses, a restful position of the limb affected, or galvanic procedures seem to have favorable effects.

Prognosis.—The prognosis is, accordingly, rather doubtful. Except in the rarer, more benignant, acute cases, the disease has a chronic course, and with exacerbations and improvements may persist for years and decades. *Permanent* improvements may occur in these chronic cases but recovery is very rare. Life is never endangered by the disease.

Supplement

Dyskinesia Angiosclerotica Intermittens

(*Intermittent Claudication and Cognate Troubles*)

This disease, though it is not actually to be considered as among the neuroses, may, for obvious reasons, be briefly discussed here. Described first by *Charcot*, later especially by Erb, it has proved to be clinically more frequent, and therefore diagnostically more important, than was at first suspected. Men of middle and especially of more advanced age are by far the most frequently affected; in younger persons and women, the disease is far rarer.

The typical picture of intermittent claudication, of the most common and longest known form of the disease, is the following: the patient who is in possession of absolutely normal motility of his legs, walks for a few minutes, a quarter of an hour, rarely longer, without any considerable disturbance; now he feels either suddenly or more gradually a pressing or drawing sensation in one or both feet and legs proper, combined with a feeling of cold and all sorts of paræsthesias, phenomena which are quickly aggravated into severe pain, forcing the patient to sit down to rest. After he has rested for a few minutes or more, he is able to walk on again undisturbed, until after a definite short time the disturbances described attack him again, and again force him to stand still.

While this typical disease process is comparatively frequent in the lower extremities, in rare cases it has been described in the upper extremities also (*Erb*, *Determann*, and others). The characteristic intermittent course of the disease is the same here also.

It is of the greatest importance that in the typical cases of dysbasia intermittens, the *pulse in the foot* concerned (either all, or only one, especially often the arteria dorsalis pedis) *is not felt at all, or is found abnormally weak.* One can usually palpate only a rigid, not a pulsating vessel. As the Roentgen examination proves, it is always a question of *arterio-sclerotic changes and narrowing of the artery concerned.* In the attack (usually to a smaller extent even in the free interval) the affected foot is of a paler more livid color than the sound foot, and has a diminished cutaneous temperature.

Etiologically, *Erb* has observed especially local cold and drenchings (in hunters, workers of the soil, etc.), excessive use of tobacco, alcoholism, less frequently syphilis.

The **pathogenesis** is still disputed. While *Charcot* and many after him explained the disease by the increasingly insufficient supply of blood—during motion and the therefore greater need of blood—due to permanent stenosis of the sclerotic vessels, *Erb*, *Oppenheim*, *Bing*, and others incline rather to the supposition of an angiospasm appearing in attacks (as a result of the comparative over-exertion). It is probable that a combination of these two hypotheses would hit the mark. In favor of the latter hypothesis is the fact that there have been observed cases in which *only in the attack* did the pulse of the afferent arteries *completely* disappear (*A. Westphal*).

In rare cases, clinicians have observed in young persons without arterio-sclerosis, a corresponding symptom complex (also with the absence of the foot pulse) (*Oppenheim, author*).

The *diagnosis, if the anamnesis is carefully taken*, is not difficult; without anamnesis it is true, the patients are often mistaken for sciatics, sufferers from gout or from rheumatism in the muscles and the joints. An exact examination of the foot pulses (which, as a matter of fact, it takes some practice to test), however, makes diagnosis easy. Occasionally differentiation from the disturbances of gait and other disturbances in diabetic or senile atheromatous sclerosis of the arteries of the legs with impending gangrene is difficult; here there are very gradual transitions to intermittent dysbasia. It must be mentioned, moreover, that the diseases are occasionally combined with neuritic or rheumatic affections of the same leg.

The **course** is always chronic and extends over years. Complete recovery is very rare, improvement is comparatively frequent. With relative frequency the patients soon succumb to other arterio-sclerotic disturbances, very frequently to coronary sclerotic angina pectoris.

The **treatment** consists of the use of local vaso-dilators especially electric foot baths. Internally, the use of preparations of iodine and nitrites is indicated. Under the influence of this treatment, I have often seen considerable functional improvement and the return of absent foot pulses. Physical rest, abstinence from tobacco and alcohol complete the therapy.

2. BASEDOW'S DISEASE

This disease was discovered by *Parry* in 1786, made known more widely by *Graves* and the Merseburg physician *Basedow* (1840), and, since 1858, following the proposal of *A. Hirsch*, has been called morbus Basedowii. Other important dates for the knowledge of the disease were the first classical portrayal by the ophthalmologist *Sattler*, 1880, and especially *Möbius's* thyroid gland theory of the year 1886.

Clinical Concept.—Basedow's disease is a general neurosis. *Its cardinal symptoms* (arranged in the order of their importance) are: *tachycardia, exophthalmus* (with the phenomena of *Gräfe, Stellwag,* and *Möbius*), *and goiter which usually pulsates;* almost as important are the *psychic changes.* usually a spontaneous and accidental increase in excitability, the *motor* disturbances usually in the form of *tremor with fine oscillations* and *muscular weakness* and the *trophic* changes, *general emaciation,* and specific *changes* *in the skin, pigment anomalies, loss of hair*, etc., and finally *secretory and dyspeptic* disturbances in the form of *hyperhidrosis* and *diarrhœs.*

The disease attacks the female sex far more frequently than it does the male; the proportion of its occurrence in the two sexes is supposedly 6 : 1; and probably even in this, the percentage is too high for the men. Children are very rarely attacked. Of importance for the occurrence of the disease are climate, that is, locality and race. The question whether so-called goitrous regions produce correspondingly more Basedow cases has been answered in the affirmative by some, while others were non-committal. After comparison of the Rhine valley in which goiters are scarce, with the Swabic in which they are plentiful, I should decide emphatically for the first point of view. Furthermore, the more temperamental and the more vasomotorily excitable races (for instance the Latin) are said to suffer less from Basedow's disease than races of the opposite type, an assertion which, however, is in need of proof. The general "neuropathic predisposition" seems, in contrast to its part in the genuine neuroses, to play a small rôle among the predisposing factors. On the other hand, in some cases (*Österreicher*, recently *Grober*) a "massed" familial appearance with pronounced heredity was observed; according to *Chvostek* and *Grober* in such families diabetes or alimentary glycosuria may alternate with Basedow's disease.

The most various things have been described as *excitant* causes. It is unquestionable that often there is no demonstrable ætiological factor. Frequently there is found long-continued over-exertion especially of a mental character, chiefly when it is accompanied by a certain undernutrition (for instance in students at conservatories, teachers). Acute psychic influences—terror, anxiety, grief—have been described as causes of Basedow's disease. *Möbius* has cleverly compared the physiognomy of the Basedow patient to petrified "crystallized terror." Sexual abstinence has been correctly emphasized by *Charcot* as a not infrequent ætiological factor. It may play a certain rôle above all in the better classes in the female sex, in the dangerous turn from "young lady" to "old maid."

Among organic causes, various acute and chronic infectious diseases, typhoid, influenza, malaria, articular rheumatism, and others have been observed; *Engel-Reimers* appeared as indorser of syphilis in Basedow ætiology. Local inflammations of the thyroid gland (purulent and non-purulent strumitis) have been regarded as forerunners or causes of the disease (*Henoch*, *de Quervain*, and others); and from these findings, far reaching conclusions have been drawn. As to the relations of pregnancy and the puerperium to the origin of Basedow's disease, statements vary. Etiologically, generative processes are certainly of no importance for the genuine disease. Möbius has correctly rejected the theory of "reflex" Basedow's disease (through diseases of the nose, etc.).

Symptomatology and Course.—The disease usually begins rather slowly, with general nervous "neurasthenic" troubles and subjective cardiac disturbances; these are followed by the first objective signs, the staring look, the symptom which often attracts the attention of the physician only, the glassy eye (*Fr. Kraus*), which is the first result of the widening of the palpebral fissure and the often striking development of the goiter in female patients; from the first phenomena to the height of the disease, weeks, months and even years may pass. Occasionally, the disease remains in this initial stage (formes frustes). An acute beginning of the disease is less common; usually then it will be a question of symptoms becoming patent through a psychical or physical traumatism or an infection, which symptoms, however, had probably slowly developed before, but remained latent. After an acute or an apparently acute beginning, the disease may run a chronic course. Cases with *acute* or subacute *beginning and course* (usually fatal, *Fr. Müller*, *West*, and others) form, fortunately, the rarest type.

The duration of the chronic form is to be counted in months and years (up to and beyond 20 years); but here it must be emphasized that we are not justified in regarding the residue of the exophthalmus, which is often left, for certain mechanical reasons, after recovery from the goiter and from the cardio-vascular symptoms, as the expression of an "active Basedow's disease."

In the **symptomatology,** by far the first place is to be assigned to the *cardio-vascular symptoms;* without them, there is, as we assume with Möbius, no Basedow's disease; without one or more of the other "classic symptoms" (cf. above) there are a great many cases of the disease. The most important circulatory symptom is *tachycardia*, which is probably always *permanent* (in contrast to the condition in other cardiac and vascular neuroses) not only accidental, emotional. Naturally this permanent acceleration of the pulse is easily capable of further increase due to motor and psychic influences. The number of pulse beats in slight and medium cases is 120–130 a minute; but frequently it is higher and may attain a frequency of 200. Arythmias are very rare, as in all *primary tachycardias of high degree*, and appear almost exclusively in the final stage. The heart is probably mostly somewhat enlarged; enlargements that are apparently very *considerable* may be traced back to the abnormally large differences (especially easily recognized by the orthodiagraphic apparatus) in the size of the heart (change in the breadth) in systole and diastole. The apex beat is correspondingly usually broadened, lying outside of the mamillary line, beating violently, but not really rising. *Schlayer* and I found the trunk of the large vessels usually strikingly broadened. In the heart there are often found systolic murmurs, especially over the base; but frequently the tones are clear, loud, and tympanic.

The large peripheral vessels (carotid, subclavian-arteries) pulsate with extraordinarily great vigor and visibility. The radial pulse, on the other hand, is usually weak, small, but somewhat rapid; the wall of the vessel is flaccid; considerably less frequent is the hardness and narrowness of the pulse, which was established by *Möbius*. The average blood pressure is not as a rule increased; the systolic pressure may be somewhat more than normal; but the diastolic is more likely to be below normal. Sphygmographically, a certain celerity in the pulse wave is displayed.

Frequently, also, there are found in the blood supply of the skin of the body alterations in the form of continuous warmth, of transitory reddening, alternating with paling, emotional erythema of the breast, dermographism, urticaria factitia, etc. In some cases I found the pletysmographic vascular reactions very much developed, especially to emotional stimuli.

The *subjective* circulatory symptoms correspond precisely to the objective ones: palpitation of the heart, feeling of oppression, annoying beating in the throat, abnormal perceptions of the pulse in the abdomen and the extremities are very common; attacks similar to those in angina pectoris (*P. Marie*) are probably rare.

All these objective and subjective cardiac phenomena may persist permanently or for a long time, without the addition of the characteristic symptoms (exophthalmus, tremor) and characterize the bulk of the *incomplete* Basedow cases.

The *eye symptoms* are doubtless the most impressive of the disease. *Exophthalmus* (the goggle eye) mostly appears slowly and symmetrically, rarely acutely; occasionally, too, it attacks the eyes irregularly. In slight cases and in the beginning, it may consist of a scarcely unæsthetic and scarcely perceptible "shining eye"; in severe cases, it reminds one horribly of the eyes of some animals. The resistance of the bulbus is not changed; pulsation in the eye ball is not demonstrable. The widening of the palpebral fissure and the abnormal rarity of winking, form the very regular *Stellwag's symptom*. Somewhat less constant but very characteristic and occasionally appearing early, is *Gräfe's sign;* in lowering the eye, the upper lid, which physiologically follows the bulbus, remains behind, and a more or less broad stripe of sclera becomes visible between the edge of the lid and the cornea. Since the symptom appears also when it is perfectly possible to close the lid, it is probably a question of a specific disturbance of the natural synergism between the movement of the lid and the bulbus. Frequently but not regularly, *Möbius's symptom* is observed; the insufficiency of the M. interni in looking at things immediately in front of the eye. But it is difficult to use this sign for early diagnosis, since slight disturbances in convergence are established as accidental findings in anomalies of refraction (myopias of even the least degree), as well as, occasionally, in some individuals, especially neuropathics.

Among rarer eye symptoms, paralyses of the external eye muscles must be mentioned (with or without paralyses of other cerebral nerves) (*Stellwag*, *Jendrassik*, and others), while the *internal* eye muscles, the dilator and sphincter iridis and their functions (contraction in light and upon convergence, dilatation in pain) nearly always remain normal. Specific sympathetic symptoms in the eye, as *Horner's* symptom-complex, are found with striking rarity in Basedow's disease. Changes in the visual field, as described by *Kast and Wilbrand* (concentric contractions) are explained by *Möbius* as the product of an accompanying hysteria. Various kinds of amblyopia do occur, but rarely. Degenerative or other changes in the disc of the optic nerve are exceptionally observed. In a very malignant case of Basedow's disease in a man, I saw atrophy of the optic nerve and total blindness. Tremor of the bulbus (analogous to the tremor of the hands) has been observed several times, occasionally associated with a vibration of the eyelids.

The lighter forms of conjunctivitis, as a consequence of insufficient protection, due to lack of covering for the bulbus and the too sluggish winking, are very frequent. More severe inflammatory or trophic disturbances of the cornea, keratitis, suppuration, even perforation of the cornea, have been reported in rare cases that were also otherwise malignant.

The **cause and pathogenesis of the exophthalmus** have been variously interpreted. Since one does not know of any muscular force that drives the bulbus forward, some authors have believed the cause of the protrusion of the eyeball to be a venous stasis in the orbits; certainly without justification, as *Landström* correctly remarks, for *such* a congestion would have to produce on the one hand, overfilling of the collateral veins (naso-frontales, supraorbitales) and, on the other hand, edema of the eyelids; but both phenomena (as permanent symptoms) are always lacking in Basedow's disease. Other authors, and indeed authoritative ones, such as *Hervieux*, *T. Kocher*, *Kraus*, and *Möbius*, have assumed an intraorbital expansion of the arteries as the cause. This hypothesis scarcely seems plausible, when we consider that arterial pulsation phenomena are always lacking in Basedow's exophthalmus, and that an intra-ocular increase in pressure is absent; *Haskovec* even established negative intra-ocular pressure in pronounced exophthalmus. We hardly need explain moreover, that the increase in retro-bulbar fatty tissue, found post mortem in certain cases, can not be the cause, but only a result of exophthalmus.

We owe to the investigations of *Landström* a very illuminating explanation of the genesis of exophthalmus. *Landström* proved that in normal eyes, the anterior part of the bulbus is surrounded as in a ring by a cylindrically arranged (up till that time unknown) muscle consisting of smooth fibres, the origin of which is in the septum orbitale, the insertion in the æquator bulbi. The contraction of this muscle must be able to cause a protrusion of the bulbus. The innervation of *Landström's* muscle was explained by the experiments of

Claude Bernard, which showed that widening of the palpebral fissure and protrusion of the bulbus could be effected by stimulation of the cervical sympathetic. *Gräfe's* and *Möbius's* symptoms are also explained in part by the action of this recently discovered muscle; in the former, *Müller's* muscle also probably plays some part.

Besides the symptoms on the part of the circulation and the eyes, *goiter* is the most constant and pathogenetically important sign of Basedow's disease. It usually develops quite slowly and regularly, and without attracting the notice of the patient, but cases of the most acute origin have also been observed. Mostly, it is only a matter of moderate enlargement, amounting to twice the size (or less) of the normal gland. Still smaller, but also essentially larger, Basedow goiters are, however, no rarity; in the latter cases, there is often a combination of an ordinary goiter with a Basedow's. The enlargement of the thyroid gland probably affects all the lobes; but assymetrical enlargement of one, especially of the right lobe, is common. Characteristic for Basedow's goiter is, above all (intra vitam) its *richness in vessels and blood;* a peculiarity which explains also the sudden and considerable fluctuations in the size of the gland. The entire goiter pulsates visibly and perceptibly, partly as a result of the expansion of the thyroid arteries, partly because of the continued pulsation of the strongly beating carotids. Over the goiter, one perceives or hears an arterial buzzing noise, occasionally also sounds from the veins (*P. Guttmann*). Subjective troubles on the part of the goiter itself are often lacking. It is, as a rule, painful neither spontaneously nor upon pressure. Pressure phenomena in the neighboring organs (trachea N. sympathicus, and N. recurrens) are probably rarer because of the softness of this goiter, than in the usual goiter, but do occur.

Anatomically and pathogenetically, Basedow's goiter has been variously interpreted. At first, clinicians considered the increase and expansion of the vessels as the one essential and the gland itself as normal. Some authors assumed a chronic inflammation, that is, the sequel of an acute non-purulent strumitis, as the substratum of the enlargement of the gland, and pointed to the supposedly frequent appearance of Basedow's disease after acute infectious diseases, as typhoid, malaria, etc. (*Roger* and *Garnier*, *de Quervain*, and others); *Engel-Reimers* has described especially an acute syphilitic strumitis with Basedow symptoms. Most probable, and anatomically the best founded, is the conception which supposes a *glandular hyperplasia* in the Basedow goiter (*S. Greenfield*, *Fr. Müller*, *McCallum*, and others). It is characterized by an enormous increase of the secretory tissue, as opposed to that of the normal tissue (analogous to the relation of the breast glands during lactation to the normal. *Greenfield*). The epithelium develops in beautiful cylindrical forms with folds in the wall, and papillary formation; rapid desquamation of the epithelium, lessening of the colloid, etc., are further typical findings. This glandular hyperplasia is found as well diffusely in the entire gland, as (more rarely), restricted to certain foci within it (*McCallum*). Similar hyperplasias have been produced experimentally by partial excision of the glands, and corresponding compensative proliferation of the part remaining. *Th. Kocher*, however, has recently opposed the theory of glandular hyperplasia, and has, on the ground of the apparently constant blood findings in Basedow's disease, a hypoleucocytosis with relative hyperlymphocytosis, pointed to the analogy of the disease and glandular swelling to pseudo-leucæmia and *Mikulicz's* disease.

Furthermore, a great number of *nervous symptoms* prevail at the height of the disease. Most striking is the well-known *very rapid*, *finely oscillating tremor of the hands*, which is often perceptible in the feet also, and rarely in the neck and head. The tremor is usually lacking in a state of rest, is most severe when the hands are extended, but increases with intentional movements (especially the fine ones), besides this also by irritation and cold. Besides the tremor, other motor irritation symptoms (for instance, choreiform movements) are quite rare; but a certain haste and abruptness characterize especially the arm and hand movements of the patient. To these described disturbances in motility, during the course of the disease and in severe cases, a few others are still added: above all, a high degree of *muscular fatigue and weakness*, especially in the lower extremities. In such cases, after brief exertions, there may be complete (intermittent) loss of strength, and sudden giving way at the knees, even a fall. These disturbances may resemble those in myasthenia pseudo-paralytica, even though they are sufficiently differentiated from them by localization, etc. Genuine, permanent Basedow paraplegias of the legs have been described also (*Charcot*). Occasionally, the Basedow paralyses are supposed to be combined with muscular atrophy. In very rare cases, also, the Basedow paralyses may be combined with manifold paralyses of cerebral nerves. The *sensory* functions are far less frequently disturbed. Sometimes one hears complaints about neuralgic pains in various nervous realms, about typical spinal irritation, paræsthesias, especially in the distal portions of the body, and the like. *Möbius* describes a frequently occurring abnormal feeling of heat without objective increase in temperature. The tendon reflexes are usually all increased; the cutaneous reflexes also may be increased. The same is true, as a rule, of the general muscular and idiomuscular excitability. The excitability of the nerve trunks on the other hand, is mostly not increased, the facialis phenomenon rarely. Disturbances of the sphincters do not belong to the picture of Basedow's disease; likewise, sexual power and menstruation rarely suffer; dysmenorrheic troubles naturally occur.

The most important nervous phenomena of Basedow's disease besides the tremor, however, lie in the *psychic realm*. Psychic changes are probably present in all, even in the lightest case. Usually they are an exact reflection of the physical-nervous stigmata: tremor and abrupt "mercurial" motility find their analogy in the psychic realm, in a persistent excitement and over-irritability of greater or less degree in an emotional respect, the tendency to sudden change of mood from the unnatural euphoria with its shouts of heavenly joy (rare) to an outburst of tears. Usually the patients are irritated by trifles, suspicious, changing quickly their likes and dislikes, forgetful, shy, moody, etc.; thus the persons, who in good health are the joy of their family, often in this disease, become its greatest distress. Naturally the fatigue often extends to the realm of the intellect, all the more, since it is increased by poor and restless sleep; the patients are no longer equal to a vocation that implies mental exertion, not even to serious reading, etc.

Real psychoses are rare in the course of Basedow's disease. On the one hand, they occur quite isolated in the final stage of the severe cases with chronic course, on the other—and that with a certain regularity—in those rare acute and malignant forms (*Fr. Müller* and others), as severe hallucinatory confusion, maniac excitement, occasionally with violent outbursts of rage, and choreiform movements. Sometimes, also, sleep conditions and in lighter cases, abnormal depth of sleep have been observed, just as in neurasthenia.

Trophic changes, especially in the skin and other epidermal tissues are common and may, in part, always be found in Basedow's disease. The skin especially of emaciated patients is often peculiarly thin, smooth and soft, usually giving the impression of an abnormal moistness; rarely it is dry. The skin of Basedow patients seems to me to resemble that of adult tetanics. This peculiarity corresponds to the lowering of the resistance of the skin to the galvanic current in Basedow patients as described by *Vigoureux* and *Chvostek*. This symptom is naturally not specific for the disease, but in no other disease have investigators found such a low stimulation threshold as in *Basedow's disease*. The cause of the phenomenon must be sought, on the one hand, in the severe soakings due to the exceedingly frequent hyperidrosis, on the other, perhaps (as recent investigations of *Otfried Müller* into the electric conductivity of the blood vessels seem to show) in the abnormal wealth of vessels, that is, in the amount of blood in the skin of these patients.

The increase in the *secretion of perspiration* is, as has been mentioned, a very frequent and pathognomonic symptom; it is found only in the hands and feet of many patients, also in the head, in others equally distributed over the entire body; it may also appear in the form of night-sweats. Unilateral hyperidrosis is rare.

Anomalies in pigmentation of the skin are very common; usually they are found in the form of dark, yellowish or brownish discoloration like that in chloasma gravidarum, in the skin of the face, in forehead, cheeks, shoulders, mammæ, navel, etc.; in rarer cases, the increase in pigment may become universal, and attain a degree like that in *Addison's* disease. Often, as in many cases of increase of pigment, places abnormally poor in pigment, vitiligo, alternate with those that are dark.

The *vaso-motor* symptoms of the skin have already been mentioned, the tendency to dermographism, urticaria factitia and emotional erythema of the face and breast. Occasionally, evanescent edema, especially in the eyelids, has been observed, less often, longer continued anasarca of the abdomen and breast or of single extremities (*Basedow*, *Möbius*). Rare complications are the combinations with scleroderma and Raynaud's disease.

A very frequent symptom that seems to me directly pathognomonic, is the *falling out of the hair* from the head, less frequently of the hair on the body. At the same time the hair is dry, lustreless and stiff; usually signs of seborrhœa are absent. The loss of hair rarely attains high degrees, probably never leads to baldness of the head, but it is a symptom that is very frequently complained of spontaneously as one of the earliest. The coarser changes in the nails are great rarities.

Among the disturbances of the digestive apparatus, there must be mentioned in the first place the *diarrhœas*, which are especially frequent even at the beginning of the disease and, under certain conditions, help to settle the differential diagnosis from hysteria with its pathognomonic constipation. According to the descriptions of *Charcot* and *Möbius*, the diarrhœas may be of various kinds: they appear either in the form of two or three thin evacuations at definite times of the day (in the morning) or, in severe cases distributed over the entire day; in the most severe cases, 30 to 40 cholera-like evacuations and sometimes rapid death were observed. Often the diarrhœas persist for weeks and months. Frequently I found in the anamneses of my patients the statement that the diarrhœas finally "stopped entirely of themselves." Usually, by no means always, the diarrhœas are rather painless. Therapeutically, it is difficult to stop them by the usual remedies. They seem to me to be most favorably influenced by rest in bed and warmth. The vomiting that has been observed in some cases is rare, and certainly but little pathognomonic for Basedow's disease. The appetite of the patients suffers considerably; occasionally, however, one finds an increase in the feeling of hunger, and corresponding voraciousness, which is in striking contrast to the lack of nutritive result, the emaciation.

The disturbances in *metabolism*, about which in recent times, a mass of literature has arisen, we can only touch upon. In the cases which end with cachexia, there is an increased disintegration of albumen. The investigations of *Fr. Müller* showed in cases, which displayed severe emaciation, in spite of the great consumption of food, and the seemingly normal digestion of the material ingested nitrogen excretions in far larger quantities than the normal (in five days 4.681g). The patients, in spite of the apparent assimilation of their food, are not maintained in a condition of nitrogenous equilibrium. *Magnus-Levy* proved an unnaturally increased output of carbonic acid, that is, an increase in gaseous interchange. But occasionally even when the evacuations contained a normal amount of nitrogen, an increase of phosphoric acid was found (*Scholz*); other authors (*Gilles de la Tourette* and others) found, on the other hand, a normal excretion of P_2O_5.

Clinically there is found in the majority of cases, a considerable reduction of the body, concerning not only the myosin but also fat and water, which is evinced in enormous loss of weight in spite of ample ingestion of food. *Mannheim*, for instance, observed in a patient a decrease in weight from 187 to 97 pounds within ten months. A decrease in weight of 20 pounds and more within a few months is found not infrequently in the anamneses of our patients.

On the other hand, there is also a not inconsiderable group of patients, who increase in weight, become even very fat. Usually they are young persons, who have the chlorotic-lymphatic habitus. But it is true, that even in these fat Basedow patients, a certain emaciation, often in the initial stage of the disease, precedes the getting fat of the patient (*Möbius*). Also during treatment, which indeed always includes the utmost rest and plentiful nourishment, one observes in spite of the further continuance of Basedow symptoms, a striking increase in weight, which far exceeds the status quo ante; there is here a striking contrast to the condition of the usual lymphatic chlorosis, in which during treatment (moderate) decrease in weight is usually established.

Glycosuria, the genuine diabetic as well as more frequently, the alimentary kind, has been observed in many cases (*Chvostek*, *Kraus*); it seems to occur specially in the so-called Basedow families and its cause has not yet been explained. Polyuria without secretion of sugar, with and without polydipsia has been observed also. A number of observations exist as to the *fever* of Basedow patients. Its frequency was emphasized but probably considerably exaggerated by *Berloge;* it appears supposedly as a transitory symptom or as a more prolonged febrile condition or may also occur as an inaugural fever. It is rather constant in the acute severe cases. Objective increases in temperature are usually lacking in spite of the increased feeling of heat in many patients, as has been already observed. A relatively high average temperature (over 100°) during the day (in spite of rest in bed) seems to me rather frequent, with a tendency to subfebrile increase, as was observed in the chloroses also.

The *respiration* of Basedow patients is usually not perceptibly changed. Occasionally, there is supposed to be a marked respiratory shallowness with an abnormally small possibility of thoracic expansion during inspiration (*Bryson's sign*). Frequently, tachypnœ is present, due partly to the subjective troubles of the heart, partly also to nervous or hysteric influences (*Reckzeh*). A typical respiratory disturbance of the Basedow patients in the form of permanent and paroxysmal changes in the respiratory curve, has been recently described by *Hofbauer.*

Subjective *serious* air hunger is at any rate a strikingly rare symptom in our patients, in spite of the often strikingly pronounced cardiac phenomena. They are accustomed to lie flat in bed, also, in contrast to patients who suffer from primary heart disease. Laryngeal disturbances are not common, and are probably conditioned mostly upon pressure lesions in the n. recurrens.

Changes in the other glandular organs (besides the thyroid gland) have been described not infrequently. Above all, in recent times, persistence of the thymus gland was found post mortem in some severe cases. The swelling of the lymphatic glands in the throat and of the tonsils is also not rare in our patients. Even cases of general lymphomatosis have been described in Basedow's disease, which show the character of Hodgkin's pseudoleucæmia, even in respect to the increase of the lymphocytosis of the blood (*Caro*). Enlargements of the spleen occur also not quite infrequently. That the liver also is implicated in the Basedow complex is proved by the observations, made by *Neusser*, of exophthalmus and tremor in atrophic cirrhosis of the liver; according to my experience, this syndrome seems to be rather frequent. The sexual organs are said, though in very rare cases, to display retrogressive changes (atrophy of the uterus, mammæ, etc.).

The osseous system is not often exposed to changes. The abnormal softness of the bones described by *Köppen* and others which reminds one of osteomalacia, is certainly just as rare as the Basedow-kyphosis described by others.

The *blood* of the Basedow patients was, until recently, considered to be altogether normal. But, according to *Th. Kocher*, it seems that, while the erythrocytes are normal in number and in their hæmoglobin content, a relative leucopenia, with absolute hyperlymphocytosis is constant in *Basedow's disease.* The time of the coagulation of the blood was found delayed, and its viscosity increased (*Kottmann*).

The **pathogenesis** of Basedow's disease is not yet wholly explained and is hotly disputed. Some theories have now only historical interest; for instance, the theory that assumed a primary disease of the heart and of the vascular system; then, that which placed the seat of the disease in the medulla oblongata (*Mendel* and others); finally, that one established by *Köben* in 1855, that local pressure of the goiter on the sympathetic caused the disease (in spite of some observations of *Fr. Müller* which seemed again to support this old theory).

It is *Möbius's* great achievement to have been the first to point out the primary rôle of the thyroid gland in the pathogenesis of *Basedow's disease.* Through the clinical recognition of the fact that the disturbances caused by removal of the thyroid glands (myxedema, etc.), formed a clear contrast to

Basedow's disease, the great empiricist reached the following conclusion: "The disease is a poisoning of the body, which arises through a *morbid activity of the thyroid gland.*" Since everything pointed to the fact that the primary change was always a disease of the thyroid gland itself, one would have to assume, *decided Möbius—that not only a hyperfunction* (*hyperthyroidization*), *but also a dys-function* (*dys-thyroidization*) *follows this disease of the glands.* That pure *hyper*-secretion is not sufficient is proved by the experience that the feeding with thyroid gland preparations can produce *only a smaller part*, and not all the thyroid symptoms, and recently the new experimental fact (*Landström* and others) that in healthy animals one can *not* succeed in evoking Basedow's disease by implanting the thyroid glands according to *Payr's* method.

The most essential support for the hyper- and dysfunction theory was given by the fundamental works of *Th. Kocher*, who on the basis of a large number of operations came to this conclusion: the more the thyroid gland tissue is eliminated through resection, the more the Basedow smyptoms retrogress; an opinion to which in 1906 *Fr. Kraus* also inclined.

The point of attack of this profuse and perverse thyroid gland secretion can, according to *Möbius's* conception, be only the sympathetic nervous system.

Then *Abadie*, *Morat*, and others reversed the matter and asserted, on the basis of experimental results, that the disease of the sympathetic (especially of the thoracalis) was the *primary* factor, since by its stimulation they thought they were able to evoke the main symptoms, exophthalmus, tachycardia, and also a congestive swelling of the thyroid gland. *Oppenheim* also, at bottom, inclines to this view, and speaks of a general neurosis, which secondarily influences especially the function of the thyroid gland. But this theory may be regarded as disproved by *Kocher's* results which prove that the partial extirpation of the Basedow goiter may cause the symptoms to disappear. For it would be absolutely incredible that the setting aside of a single (supposedly) partial resultant phenomenon of the condition of sympathetic irritation, among many others, should in itself end this condition of irritation. Thus the primary agent of the disease must be sought in the thyroid gland itself, and only the secondary in the irritation condition of the sympathetic, the existence of which, on the other hand, as I shall state in advance, *Landström's* pioneer investigations verify.

In sharp contrast to *Möbius-Kocher's* theory of hyper- and dys-secretion, stands that of *v. Cyon and especially Blum*, who look upon the normal rôle of the thyroid gland as an "*unpoisoning*" of the organism, a ridding it of autotoxic and other products, and upon *Basedow's disease* as a disturbance in this poison removing process. *Blum* especially has drawn clearly this negative picture of *Möbius's* theory, and attempted to support it by a series of experimental investigations; he says: the thyroid gland secretes nothing at all, not even any vital toxin, but within it poison removing processes take place; the matter from which the poison must be removed, against which physiologically the organism must be protected, originates mostly from the meat ingested, and from the endotoxins formed from it in the intestines by metabolic and bacterial processes.

Blum, then, does not accept hypersecretion as the primary factor in the origin of Basedow's disease, but an insufficiency in the physiological poison-removing capacity of the thyroid gland, sharply rejecting *Möbius's* theory. He sees the main poison removing substance of the gland in its iodine. Logically therefore, in the treatment of Basedow patients, he employs first iodine, and secondly, abstinence from meat, and condemns operative treatment in spite of its glorious successes.

Fr. Kraus in his Munich report, 1906, has pointed out, correctly, that the diametric contrast between the secretion and poison-removing theories, which has been constructed by those authors, is by no means absolute. He assumes that there is a typical substance secreted by the thyroid gland but also emphasizes the possibility that the gland, in addition to producing substances which are of importance for the persistence and functioning of the organism, or of single organs (inner secretion in the narrower sense) produces also some which serve especially for the removal of poison from certain injurious metabolic products, from other organs, or from exogenous poisons (for instance, atropin). Why should not the function of a *secretion* consist also in this *removal of poison?*

I should with *Landström*, especially because of the overwhelming success of *Kocher's* operations, adopt above all the hyper- and dys-secretion theory of *Möbius-Kocher*.

The way by which the increased and perverse secretion (or according to *Blum*, *v. Cyon*, and others, the endotoxin whose poison has not been removed) creates the Basedow complex, can only be by the sympathetic nervous system. The nervous secretory, vasomotor and trophic symptoms were long since obviously interpreted as sympathetic disturbances. Only the eye symptoms did not fit in with this explanation. Now the chain of proof is made complete by the discovery of a smooth fibred orbicular muscle, therefore subject to the sympathetic, which fully explains the exophthalmus and the other eye symptoms (*Landström*).

So we come to the same conclusion, that *L. R. Müller* also reached in his discussion on the pathology of the sympathetic in this book: Basedow's disease is *no primary* general neurosis, no primary neurosis of the sympathetic, but a *secondary neurosis, the nature of which must be viewed as a chronic autointoxication through the increased and, as to its condition, essentially changed secretion of the thyroid gland and its influence on the sympathetic nervous system.*

The **differential diagnosis** of Basedow's disease need be discussed but briefly. The developed system complex of the "goggle eye disease" is so characteristic that it can be confused with no other disease. The incipient and permanently incomplete forms of the disease present more difficulty; it is often not easy, occasionally impossible, to differentiate them from a primary vasomotor neurosis in neurasthenia, or (more rarely) hysteria. This is especially true of that vascular and cardiac neurosis (already mentioned in the discussion of vasoconstrictor neurosis) of young persons, which runs its course with a pronounced tendency to vasodilatation (emotional erythema, erythrophobia, dermographism) and to the "cor juvenum." But I hold with *Oppenheim* that in such cases the *permanence* of the tachycardia is the decisive criterion. If one examines frequently the simple neuroses in a state of rest, one will find that the tachycardia that was originally psychically evoked, ceases, and gives way to a normal pulse, while in Basedow's disease the tachycardia persists continuously, even though it is usually capable of further emotional increase.

Certainly many of the other symptoms can be simulated by hysteria—the psychogenous diarrhœas, the anorexia and voracity, even a high degree of emaciation (as a result of open or secret abstinence of food). But if to such symptoms there be added a permanent tachycardia of 120 or more pulsations a minute, one always thinks of an incipient or incomplete Basedow's disease. Such types of not infrequent occurrence are the combination of tachycardia with persisting diarrhœas, or with loss of hair, or with sweating and changes in the pigmentation, or finally with an isolated tremor of the hands, etc.

It is important, furthermore, that in simple goiter, cardiac symptoms of a peculiar kind are said to appear, which owe their origin to various influences (*Minnich, Fr. Kraus*). But the "goiter-heart" has not a few traits in common with the Basedow heart, so that one may wonder whether they are not different degrees of *one and the same* affection (hyper- and dys-thyroidization). How can one clinically draw a sure line between a forme fruste of Basedow's disease with predominance of the circulatory symptoms and a goiter heart? That goiters in a purely mechanical way, by pressure on the vagus, veins and trachea, may cause certain cardiac symptoms, should not in itself be interpreted as a specific result of the *thyroid gland tumor*. In uterine myomatosis a condition has also been observed not unlike the goiter heart (so-called myoma heart).

Treatment.—It is evident that with the wide divergence of the etiological and pathogenetic conceptions, the therapeutic views must diverge also. We should mention first as a very important factor, the necessity of the *absolute removal* of the patient *from his vocation* (even in the light incomplete forms); ambulant treatment (which occasionally may be effective in some other neuroses) will probably always fail in Basedow's disease.

For patients of less means, the hospital or sanatorium is the suitable place. Treatment by rest in the open air will, as in the treatment of tuberculosis, be very beneficial. Severe cases naturally should be confined to bed. We should advise the well-to-do patients to stay at a resort where they may benefit from the *mountain air*, presupposing naturally that their condition is not so severe as to demand *clinical* supervision. According to the constitution of the patient, moderate altitudes or even sheltered places in the high mountains are recommended, localities 1200 to 1700 m. in height. From residence at the latter (St. Moritz, Madonna di Campiglio and others) when the patients stay there for a long time, very good results are attained, especially with regard to subjective welfare.

As to the diet, clinicians are agreed to-day, that an unstimulating diet, with little meat and containing rather the carbohydrates, fat, milk, vegetables, etc., is the right one for the patients; this is the easiest way, comparatively, of increasing the weight of the patients. Highly seasoned vegetables, sour things, especially alcoholics and strong tea and coffee should be restricted or entirely forbidden, as well as the use of tobacco.

Naturally one must adapt one's self in all this, to the peculiarities of the patients, to their inclinations and aversions, and consider especially their tendency to diarrhœa (from the most heterogeneous things); for instance, one must frequently reckon with the fact that milk is absolutely not digested.

On the basis of his experimental and clinical experience, *Blum* (cf. above) has recommended a continuous vegetable diet, if possible entirely without meat. I do not believe that this rigorous diet, which has found only few adherents, is advisable; in practice I have never found it successful.

Of *hydro-therapeutic* measures, baths containing carbonic acid, or other substances (extract of pine needles, salt, etc.) are recommended, also massage, sitz-baths, etc.; usually the effect of these procedures is more of a symptomatic or even of a suggestive nature.

The same is probably true of *electro-therapy* also, though we must point out that the stabile galvanization of the cervical sympathetic, the most authoritative method, enjoys the approval even of modern clinicians, for instance, *Fr. Kraus;* this latter procedure then is certainly worth trying in cases that are treated in an institution.

In respect to *medicaments*, we would suggest at once the ideal tonic, *arsenic*, which in internal (or even better) in subcutaneous administration, for instance as natr. cacodylicum (sodium cacodylate) without doubt, frequently gives good results; the regulation of the doses of the arsenic should follow the usual custom; above all the dose should be small at first and gradually increased. Some recommend preparations of phosphorus of various kinds. The same is true of quinine. Remedies which have a specific influence on the nerves, atropin and ergotin have been recommended, but are not very popular. There are various views as to the indication of iodine, explained partly by the differing pathogenetic views of authors.

Those that defend the hypersecretion theory, must naturally condemn the iodine as a considerable component of the secretion of the thyroid glands and renounce treatment with iodine (*Möbius*). Also, *Kraus*, *Landström*, and many other clinicians deny that iodine is of any service therapeutically. Other authors (*Kocher*, *Strümpell*, and others) recommend under certain conditions small doses to prepare for the operation. *Blum*, from his point of view, naturally is an energetic defender of treatment with iodine.

Concerning **symptomatic** treatment, it will often be necessary to give sedatives to excited, sleepless patients, which will frequently prove to be effective, such as bromine, valerian, in the various old and new forms in which they are given; in more severe cases, soporifics, such as trional, veronal, paraldehyde, and other similar remedies. The cardiac symptoms, above all, the tachycardia, unfortunately can not be influenced by the usual cardiacs, digitalis and strophanthus; therefore they need not even be tried. Naturally, in very severe cases, with typical signs of failing *compensation* of the heart, they are again indicated. The diarrhœas are, as has been remarked, often refractory to opium and astringents; rest in bed, warmth, and diet are of more service.

The internalists hoped for great results from the treatment of Basedow's disease by *specific antitoxins*. The best known remedy of this kind is antithyroidin serum *Möbius* (prepared by *Merk*) which is given in doses increased from 0.2 to 2.0 and even 5.0. It is extracted from the serum of the blood of sheep whose thyroid glands have been removed. The point in this treatment is that the toxic substances that appear in the blood of these athyroid animals, would be apt to neutralize the excessively produced secretion in individuals who suffer from an excess of thyroid secretion (hyperthyroidism). Besides *Möbius's* remedy, there has been recently recommended rodagen, a preparation from the milk of goats, whose thyroid glands have been removed (*Lanz*). The results of this serum, that is, antibody treatment, were much extolled by authors, especially in former years. But there have been also sceptical critics (*Strümpell*, *Eulenburg*) and it almost seems as if the great wave of surgical treatment were about to sweep away this treatment also.

Of historical interest only are the practically useless and theoretically unfounded attempts to treat Basedow's disease with preparations of the thyroid glands.

The results observed from the administration of organo-therapeutic preparations of the thymus and ovary (*Owen*, *Miculicz*, *Dalaunay*, *Oppenheim*, and others) are very interesting; they originate from the principle that these glands of internal secretion may vicariously substitute each other, as has recently, with greater and greater probability, proved to be the case.

We are however more and more forced to confess that no methods of internal treatment can boast of having attained such constant and permanent results as surgical treatment: *partial strumectomy*, whose most successful defender is *Th. Kocher.* Without entering upon a description of the various methods, past and present, I shall discuss only that of *Kocher;* it consists in a preparatory, general strengthening treatment for several weeks (with natr. phosphor.) and later in partial strumectomy (usually performed several times) combined with ligature of the arteries; if possible one should operate under local anæsthesia and avoid narcosis. In case one operation is not successful, a repetition is recommended. Surgeons have long since of course desisted from a total extirpation of the goiter, which causes first, a cachexia strumipriva, and secondly through the frequently resulting removal of the parathyroid glands, tetany. The results of surgical treatment, in spite of some sceptics even among the surgeons themselves (Garré), have improved from year to year. We must emphasize especially that *Möbius* greeted and recommended it enthusiastically as a confirmation of his theory. Internal clinicians also (*Fr. Kraus*) and neurologists have become adherents of surgical treatment, while others (for instance *Strümpell*) are still reserving that judgment. *Kocher's* results are, however, so great and convincing that all opposition will soon die out.

As early as 1906, he could report 167 cases of Basedow's disease treated by operation, of which 93% were cured or were considerably improved; the mortality amounted to only 5.3%. Two years later, Kocher published 153 more cases, among which he could establish easy and complete cure in 70%; the mortality in this series, moreover, comprised only two cases. *Kocher*, then, was justified in saying, that in the hands of the skilled operator, the extirpation of a Basedow goiter was not much more dangerous than that of an ordinary goiter. Of more recent results that confirm these we shall mention only those of *Landström*, who in a large number of cases observed cure or great improvement in 71%. Concerning the interesting details of the course of recovery after the operation, I refer to the descriptions of *A. Kocher and Landström.* It must merely be mentioned that the latter frequently observed post-operative states that gave the impression of acute intoxications.

Other methods supposedly serving to cause a diminution or involution of the thyroid gland, like electropuncture, injection of iodoform, etc., can not be regarded any longer as being up to date. The attempts, too, to diminish the Basedow goiter by X-rays (*Pusey* and others) have apparently no future, judging from the negative results of *Pfeifer* and *P. Krause.* Finally, the resection of the sympathetic (*Jonnesco, Abadie*) which seemed justifiable on the ground of the already mentioned pathogenetic theory of the latter must be considered. The results of these authors, however, have not stood the test of others; *Landström* recently declared the results of the resection of the sympathetic to be very doubtful.

The **prognosis** of Basedow's disease must, as is evident from the above discussion, be variously formulated to-day, according as to whether the patient does or does not submit to surgical treatment. There is no doubt that many light, and even moderately severe cases, and especially the incomplete ones—above all, if the patients belong to the wealthy classes, in which much time and money can be spent upon climatic and other treatment, may be cured even without an operation, by internal treatment. It is true that such recovery always occurs slowly, in several months, even in years. At the same time it is rarely a question of actual total recovery, in a scientific sense, since one or another symptom, even though a slight disturbance in itself remains as a trace; thus occasionally a residue of exophthalmus, or light tremor, or finally a tendency to tachycardia may remain. In very rare cases, one must reckon with the transition of Basedow's disease into its opposite, myxedema. The mortality of Basedow's disease fluctuates according to the statements in the literature, between 10 and 25%; I believe that these figures are too high, and take into too little consideration the numerous harmless, slight and incomplete cases (especially in the wealthy classes). Finally, these figures will have to be lowered, when surgical treatment becomes still more widely spread, for even severe, the most severe cases have not infrequently been cured by strumectomy. Nevertheless, great emphasis must be laid on the words that Kocher wrote to the congress for internal medicine in Vienna "Do *not* send us the Basedow patients for operation when it is *too late*."

(3) MYXEDEMA

(a) Myxedema of Adults (Cachexie Pachydermique)

Spontaneous, as well as post-operative myxedema, may be designated as the *direct converse to Basedow's disease*, in pathogenetic and in many ways also in clinical respects: while Basedow's disease, according to *Möbius-Kocher*, must be interpreted as a specifically *thyreotoxic* condition, it is a question here of a *thyreoprival* symptom complex.

Myxedema was described first by *W. Gull* (1873); *Hadden* first pointed out the ætiological connection of the diseases with the thyroid glands. Among German authors, *Virchow*, *Ewald*, *Kocher*, *Magnus-Levy*, *Scholz* and others must be mentioned as pioneers in the pathology of the disease.

Clinical Concept.—By myxedema we understand a cachexia which begins spontaneously in middle (or more advanced) life and which usually progresses with an uncommonly chronic course, the most striking symptom of which is a peculiar (pachydermic) thickening of the skin (especially in the face and the extremities), and general physical and mental decay; we regard as the cause of the disease, as has been mentioned, a functional disturbance, namely, destruction of the thyroid glands.

Etiology and Pathogenesis.—As exciting causes of myxedema various noxæ have been described; above all, excitement, worry, and other psychic traumatisms, rapidly recurring parturitions, severe hemorrhages (*Magnus-Levy*), infectious diseases (most frequently erysipelas, influenza, dysentery, and others); syphilis has been also ætiologically accused. That hereditary factors may be of import is shown by observations of familial myxedema adultorum (*Putnam*, *W. Ord*); as to the significance of familial predisposition to tuberculosis for the etiology of our disease opinions of the authors vary; *Pel* speaks for it, *Magnus Levy* against it.

All authors agree on the *pathogenetic rôle of the diseased thyroid glands* for myxedema, in spite of the fact that the nature and real character of the disease is still obscure. In a pathologic-anatomical respect, it is a matter, according to *Ewald*, of a more or less high degree of atrophy of the thyroid gland. This is described in almost all post mortem cases, as diminished in toto, often as "hard, fibrous, of yellowish white color." Microscopically, there appears considerable proliferation of the interalveolar connective tissue, which causes destruction of the glandular parenchyma. The (rare) enlargement of the myxedematic thyroid gland has been traced back, as a rule, to strumous growth of the interstitium, and not to increase of the glandular tissue.

It would take too long to enter here on the histology of the skin. An interesting finding was made in the form of an (apparently vicarious) enlargement of the hypophysis in myxedema (*Ponfick*).

Symptomatology and Course.—The disease on the whole is very rare, apparently more common in England and Belgium than in Germany. Countries in which endemic cretinism is of frequent occurrence have apparently no strikingly high myxedema morbidity (*Magnus-Levy*). It attacks far more women than men (proportionate morbidity 4:1, *Ewald*) preferably married women and mothers to unmarried girls. The beginning is usually extremely slow and insidious; the initial stage with its obscure symptoms may extend over many months, even over years, until finally the appearance of the classical changes makes diagnosis possible, but cases have also been described in which, for instance after an exhausting disease, the affection appeared rather acutely. As a rule, the disease begins with quite general disturbances: a slow physical and psychical retrogression, manifold symptoms of nervous, hysteric and even vasomotor type. Then, sooner or later there begins—occasionally after an eczema or erysipelas—the change in the skin, especially in the face; there gradually comes to be a peculiar elastic edematous swelling of the eyelids, cheeks, lips, nose, ears, in short, of the skin all over the face. The swelling is not suffused and hot, but usually pale, occasionally like porcelain; it is not soft and doughy, like cardiac or renal hydrops, but harder and stiffer; pressure of the finger does not result in pitting. In contrast to the skin of sclerodermics, the skin of myxedema patients is often easily movable upon the underlying parts, often abnormally movable.

At the height of the disease, the cheeks hang down like "cheek-pouches," the region of the lower lid forms plump sacks, the eyes become narrow slits, the upper lid protrudes snout-like, the forehead becomes wrinkled into immovable hard folds. This plump head that must look something like a dropsical Eskimo's, is placed on a likewise plump short neck, on the front and side of which swellings occasionally appear. The hands, too, are early attacked; they become plump, bloated, finally swollen high, like "gauntlets" (*Ewald*). At the same time, they increase only in breadth and thickness, in contradistinction to acromegaly; the nails—also in contradistinction to the latter disease—are said to frequently suffer changes (become brittle, fall out). In the feet there are found changes in the skin analogous to those in the hands. The skin of the body is also affected, though rarely to the same extent as in the head and extremities; the trunk may thereby become plump and barrel-shaped. The skin shows the following remarkable condition, directly contrary to those of a Basedow patient: it is not only thickened, but usually also excessively dry; often the temperature of the skin is lowered; there is also mostly a subjective feeling of cold. The resistance of the myxedematic skin to electric conductivity was by some authors found increased.

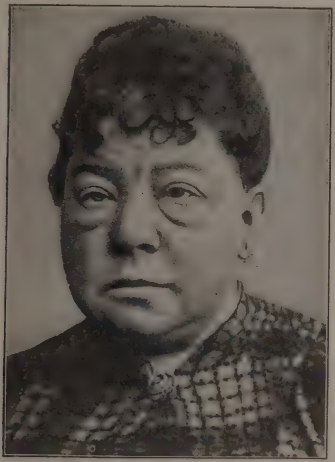


FIG. 226.

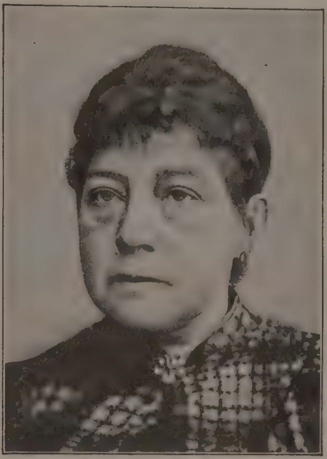


FIG. 227.

FIGS. 226 and 227.—Myxedema. Before the treatment. After the treatment. (*From Magnus-Levy.*)

The hair of the head, often too, that of the body, falls out and becomes sparse, occasionally discolored. In individual cases abnormal hair supply (woman's beard) was found. The mucous membranes of the mouth, tongue, pharynx and larynx also, are usually not spared by the pachydermia; but exceptions to this have not infrequently been observed.

With the increase of the pachydermic change in the skin, the entire physical man is changed: the movements (above all the gait) become slower and clumsier; the gross strength decreases (though there is no direct paralysis), the musculature becomes atrophic; the patients in severe cases finally become invalids and helpless cripples. The mental decay frequently (not always) progresses still more rapidly than the physical. *The patients suffer mental impoverishment;* they become dull, devoid of interest and energy, their emotional excitability is lowered; nearly always there is a depressed mood; memory decreases also, as does the power of judgment. This simple decrease of psychic and intellectual power, is the most common picture of the psychic disturbance; it is less often mixed with hallucinatory confusion, and manic or melancholy symptoms; but apparently typical myxedema psychoses have also been described. In rare cases, however, in spite of a high degree of physical changes, there may be no disturbance in intellect at all (*Magnus-Levy*). Of subjective phenomena we must mention furthermore, the very frequent headaches and vertigo. On the part of the cerebral nerves, an impairment in hearing is frequent (*Oppenheim*). Changes in the optic nerve were observed in isolated cases; disturbances of the other cerebral nerves are certainly extremely rare. Changes in speech seem to occur almost regularly; the voice becomes rough, deep, and monotonous; articulation may suffer also (stumbling over syllables).

The sensibility except for the subjective and objective feeling of cold, is usually not disturbed. Except for the general difficulty in movements, real motor changes are very rare; in some cases, tremor of the hands has been observed. We must mention here tetany (*Kräpelin* and others) as an interesting, but in consideration of the genesis of myxedema, extremely rare syndrome. It may be combined with epilepsy in the form of petit mal. The tendon reflexes are usually described as normal, occasionally as lowered.

Of the internal organs, we must think first of the *thyroid gland.* It is in the majority of cases described as small or even altogether lacking; if one considers how difficult and uncertain it is to establish the size and form of the normal gland by palpation, one must take these statements a little sceptically. Occasionally, the gland has been found perceptibly enlarged, and strikingly hard.

Of the other organs, the *kidneys* have been found injured with relative frequency. Albuminuria and cylindruria form a rather frequent syndrome of the disease. The *heart and vascular system* suffer rarely, probably mostly in those cases in which a myxedema has supervened upon a Basedowoid disease of the thyroid glands. In late stages of the disease there are, naturally, as in all cachexias, phenomena of cardiac insufficiency. Rather frequently one observes in myxedema symptoms of hemorrhagic diathesis. Of *metabolic* disturbances, glycosuria is to be mentioned (*Ewald*); it seems, however, to be very rare. Exact investigations of the *gaseous changes* in the various forms of hypothyroidism (*Magnus-Levy*) showed that all severe cases exhibit a high degree of *lowering* of gaseous interchange, to about 50 to 60% of the amount observed in healthy persons; again a most characteristic contrast to Basedow's disease, in which a considerable increase of the same was observed. This decrease is supposed to be dependent not upon a smaller quantity of functioning protoplasm, but upon its slighter vital energy. The appetite of the patient is usually decreased, the bowels sluggish. At the height of the disease, the weight of the body is always very considerably increased. Alterations in *activity of sexual organs* are common in our patients; in women menorrhagias are supposed often to occur; by some authors, however, the opposite is asserted. Frigidity is also observed. In men, rapidly appearing decrease and loss of sexual power was found.

In respect to the motor apparatus we have a not infrequent chronic synovitis of the knee joint—and in younger patients—changes in the growth of the bones.

Differential diagnosis of the spontaneous myxedema of adults is usually not difficult as soon as the typical changes in the skin have appeared. In the stage that often precedes—that of slow general physical and psychical reduction—the diagnosis is naturally most obscure.

At the height of the disease, the following conditions are to be considered in differential diagnosis; above all chronic erysipelas of the nose and the lips, which with a non-febrile course and with perpetual external exacerbations, may simulate a forme fruste of myxedema, but in which cachexia and psychic decline are completely lacking. The same is true of the evanescent edemata of the climacteric period and of syphilitic pseudo-myxedema (*Oppenheim*). Even the—very rarely frankly expressed—stage of hard edema in scleroderma, has only superficial resemblance to myxedema which is detected by palpation, and furthermore, by consideration of the other myxedemic phenomena. That in dementia præcox a habitus may appear not greatly different from myxedema (*Meige and Dide*) I can confirm from my own experience. The various kinds of pathological accumulation of fat (*Dercum's* adipositas dolorosa, lipomatosis perimuscularis) (*Heinr. Curschmann* and others) may display a similarity—though only a superficial one—to myxedema; it should be mentioned, moreover, that by many authors, *Dercum's* disease is also interpreted as a hypothyroidism.

(b) Cachexia Strumipriva

represents a disease that is extremely similar to spontaneous myxedema, even in its nature, and which is distinguished from it only by the fact that its cause (here easily recognized) is a *sudden* destruction of the function of the thyroid glands by a local strumectomy, and that the thyreoprival cachexia correspondingly appears somewhat more quickly.

The credit of having first recognized this most important sequela to local removal of the thyroid glands and of having perceived its essential likeness to myxedema, is due to *Th. Kocher* (1883); *Reverdin* has also been of service in the development of the pathology of the disease, likewise to a great extent the London myxedema commission (1888).

Postoperative myxedema was not so very rare before surgeons had learned the severe consequences of total strumectomy (just like strumiprival tetany); now it has probably become extremely rare. It was naturally observed most frequently in goitrous regions and in those predisposed to goiter (women).

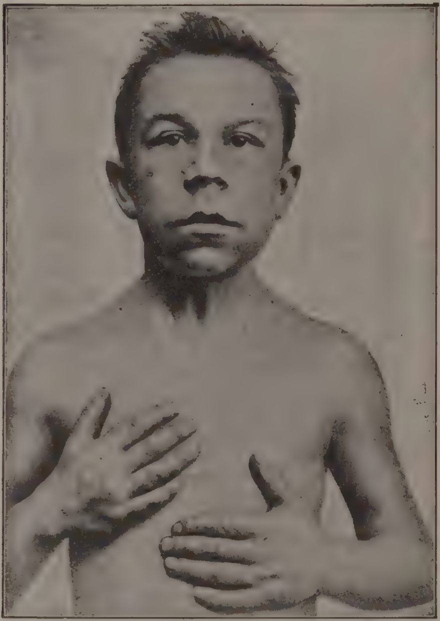


FIG. 228.—Cachexia strumipriva in a 28-years-old man (before the treatment). (*From Magnus-Levy.*)

Soon after the strumectomy—one or more weeks afterwards, less frequently even after months and years—the patients suffer from quite general symptoms; heaviness and fatigue in the limbs, weakness, trembling, feeling of cold and similar phenomena. To these there is added sooner or later a gradual decrease of the psychic functions; mental activity, interest in the vocation, memory decline. The patients become mentally deficient. The younger the person affected, the more severe usually the mental and physical phenomena of functional loss are. Simultaneously changes in the skin analogous to those in myxedema appear, with especial severity in the face, hands, and feet; the hair of the head and body falls out. At the same time —not always—there appears a general corpulence. The genital organs remain infantile, if the patient who has undergone strumectomy had not reached puberty, as in the classic case of *Magnus-Levy*. Likewise in these patients the entire growth remains at the stage of childhood. This is especially characteristic in the development of the bones; the epiphyseal cartilage structure remains unchanged and in a person 30 years old may create the impression of a child of 14. The mental development also of these individuals stops altogether at the stage of childhood, so that they usually can not follow any vocation whatever (Fig. 228).

Further symptoms of myxedema are anemia, which seems to consist usually in an equal decrease in the number of erythrocytes and in the hæmoglobin content, and certain symptoms of cardiac weakness (small pulse, occasionally dilatation of the heart). Less frequent phenomena are the narrowing of the larynx, which has even given occasion for tracheotomies (*Baumgärtner*), a peculiar retardation in respiration (to 6 respirations a minute) and tetanic and epileptic attacks. *Pineles* has shown that tetany appears with varying frequency according to the operative technique employed.

Not all patients who have apparently suffered total strumectomy are attacked by myxedema. Observers have therefore concluded that in these cases (in which no tetany appeared either) the accessory thyroid glands vicariously assumed the function of the main gland. Cases have also been observed in which the myxedema attained only slight degrees, in which the psychic effects were minimal, or cases in which symptom complexes at first severe later retrogressed spontaneously (myxedeme fruste). In some of these cases, the cause of this lack of progress or of the spontaneous improvement, was proved to be the development of a little bit of goiter left behind in the operation by mistake (*Reverdin*).

Incidentally it must be mentioned that even after partial strumectomy, myxedema has been occasionally observed; as cause of this phenomenon, Kocher found post-operative atrophy in the *non*-extirpated parts of the gland.

Concerning the rest of the symptomatology, metabolism, etc., we would point to the discussion of these things in spontaneous myxedema; in these respects there are no differences between the two forms of the disease. In respect to differential diagnosis also of postoperative myxedema we need add nothing to what has already been said.

The **prognosis** of well developed spontaneous myxedema adultorum is, if not skilfully attacked therapeutically, usually unfavorable. After an illness of many years, the patients succumb either to cachexia itself or to intercurrent diseases. In operative myxedema, the chances seem to be a little better; here, as we have seen, spontaneous standstills and long remissions have occasionally been observed; formerly, in most cases, it is true, this form also ended in death after 4 or 5 years (*Ewald*).

The causal **therapy** of myxedema has very considerably improved this unfavorable prognosis, transforming it in the majority of cases even to a favorable one quoad vitam.

It is evident that the treatment must consist in a specific organo-therapy to compensate for the defect, that is, in the administration of thyroid gland substance. It would take too long to exhaust the many varieties of this treatment; suffice it to mention here the most tried and essential. The simplest form of administration, to give the animal thyroid glands in raw or cooked condition (per os or even per rectum) has, it is true, attained definite results, but has not persisted because of many disadvantages (easy putrefaction, bad taste, confusion with other glands). Another theoretically ideal substitution of glands would be the implantation of a sound organ (if possible of like species).

It was first tried by *Th. Kocher* (in the throat), later by *Schiff*, *Bircher*, *Horsley* and others (mostly in the abdominal cavity), partly with good, partly with negative results. Recently *Payr* has undertaken implantation into the spleen and *A. Kocher* into the marrow of the bones (epiphyses); the latter modification is pronounced by *Th. Kocher* to be very promising.

Nevertheless, for the present, the most facile and harmless treatment is that with the organo-therapeutic preparations offered. Especially popular are above all, the thyroid tablets of *Borrough, Wellcome & Co.* (dried mutton thyroid glands, furthermore including parathyroid glands, *Pineles*) each of which contain 0.33 gr. of thyroid gland substance; but the German tablets (Cologne, Dresden) have been quite as effective according to my own experience. One gives adults 1 to 4 tablets daily, increasing the amount slowly, and to children first one-third to one-half tablet (that is 0.1 to 0.15 gr.) once or twice pro die, increasing gradually to a maximum of 2 tablets (careful gradation). The "thyrad tablets" prepared according to Kocher's method have also been praised.

Iodothyrine (tablets) and thyreoglobulin of *Oswald* (containing 1.66% of iodine) are, according to *Magnus-Levy* quite as useful as the *Kocher* tablets. *Fränkel's* thyreoantitoxin is praised; but the latter preparations have the disadvantage of being very high-priced. The same is true also of the (apparently very different) thyreoproteid of *Notkin*. Besides these, various inorganic and organic preparations of iodine, hypophysis and thymus tablets and others have been used and recommended.

The main thing in organo-therapeutics is a *lasting*, but always *careful* procedure. One should give the tablets or other preparations according to their results continuously for many months—interpolating brief pauses—if necessary a whole year and even longer. At the same time one must consider that there are some people with idiosyncrasies that react against the smallest doses (appearance of tachycardia, agitation, nausea, syncope), and on the other hand, during the course of the treatment, intoxication phenomena of the same kind may gradually appear. On the whole, the latter cases are very rare when the doses are very cautiously increased. It is recommended finally, once the cure has taken place, just as in *Kussmaul's* digitalis treatment, to continue small doses for a long time as a prophylactic measure.

Other non-specific (possibly tonic or diuretic) medicines need no special mention.

Concerning the *effect* of organo-therapeutic preparations, there frequently appears, even a few days after the beginning, a change in the activity of the skin, retrogression of the sweat secretion and normal temperature of the skin, decrease in the accumulation of fat and at the same time, considerable decrease in weight (for instance 22 pounds in 4 weeks, *Magnus-Levy*), normal metabolism, return of the hair growth, and above all psychic restoration; the patients are physically and mentally as if "newly born." In young patients there follows (even beyond 20 years) renewed growth of the bones (disappearance of the intervening cartilage at the epiphyses), growth of the beard and normal sexual development in place of the infantilism; a patient 28 years old of *Magnus-Levy* could, after 2 years' treatment, boast of a moustache and—gonorrhœa.

Adipositas dolorosa (*Dercum's* disease). Here we may refer briefly to the quite rare disease described by *Dercum* in 1892: in women of middle or more advanced age: (most frequently during the climacteric period), very rarely in men, there is a considerable general or even circumscribed lipoma-like accumulation of fat which attacks especially the trunk, nape, and upper divisions of the extremities, almost never the face and the hands; these accumulations of fat are peculiar in being extremely *painful*, spontaneously as well as upon pressure. The disease may be combined with constitutional anomalies and—apparently very frequently—with psycho-neuroses. In some cases, retrogressive *changes* have been found *in the thyroid gland*, so that many authors describe the disease as thyreogenous, and assume a certain connection with myxedema. With *Oppenheim* I should warn against diagnosing this disease too easily, as has heretofore been the case. The syndrome adipositas and genuine and climacteric hysteria is so enormously common that there is danger in assuming the presence of Dercum's disease far too frequently in such patients on the base of hysteric neuralgias, hyperalgic zones, etc. Adipositas dolorosa has undoubtedly close connection with symmetrical lipomatosis of various kinds in men, which also in statu nascendi may be accompanied by pains. That in such cases, also, there might be association with the thyroid gland I saw from a case which was combined with Basedow symptoms and pigmentations.

(c) Infantile Myxedema and Cretinism

The theory of endemic and sporadic cretinism and its relation to myxedema has always been the subject of eager controversies; the conceptions have been very varied. In the following I am keeping close by the statements of *Friedrich Pineles*, who, in my opinion, has investigated and arranged the material on the basis of anatomic, clinical and experimental investigation with great judgment and thoroughness. We distinguish with *Pineles* in the symptomatic picture of *sporadic cretinism* two forms: 1. *thyreoaplasia, congenital myxedema; 2. infantile myxedema.*

1. Thyreoaplasia Congenita

As to its appearance the disease is confined neither to goitrous nor to myxedemic regions, but occurs *everywhere*. The female sex is supposed to be far more frequently attacked than the male. Concerning heredity in specific or other respects, the statements vary considerably; but it seems that besides the excessively noted "neuropathic predisposition," tuberculosis and consanguinity, no "taint," especially of goitrous diseases, is usually present. The course is such that children are apparently born normal, and (according to the opinion of the mother) develop normally; but the experienced physician may in such cases even in the first months recognize the incipient myxedema. Only at the age of half a year or later do the striking and coarse disturbances begin; the physical as well as the psychic development halts. The children who were cheerful and lively and had possibly already made attempts to walk and talk, lose the progress made. Gradually the picture of the myxedematoid cretin develops; the skin becomes thick but usually spongy, the lips are swollen, the nose broadened, the eyes small and slit-like, owing to the swelling of the lids. Besides this, there are ridges of fat, especially on the sides of the neck, the abdomen is fat and there is frequently an umbilical hernia. The growth of the bones is retarded, the fontanelles remain open abnormally long, the intervening cartilage at the epiphyses persists even beyond twenty years (*Scholz*). Since actual changes (except for the hypoplasia) are lacking in the bones, the plumpness

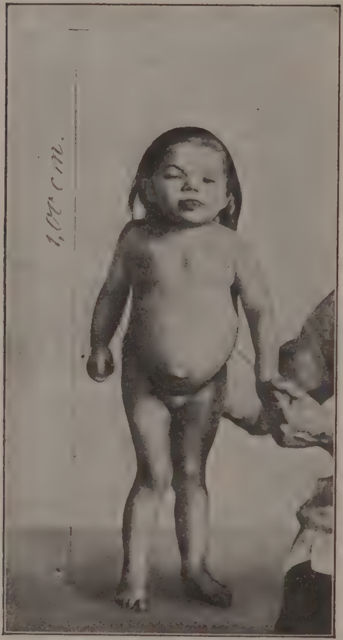


FIG. 229.—Sporadic cretinism (thyreoaplasia congenita). Girl, 14 years old, before the treatment.

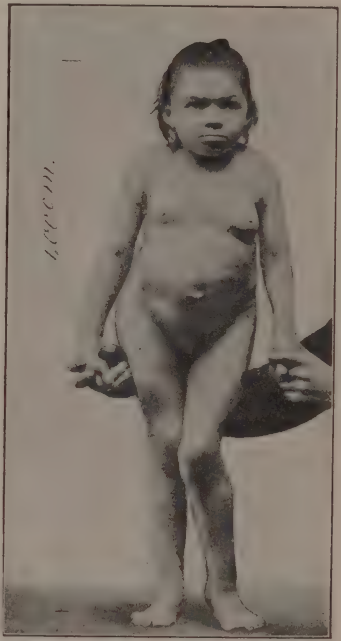


FIG. 230.—The same case after 4 years' treatment. (*From Magnus-Levy.*)

of the extremities rests upon the increase in the tissue of the soft parts. An abnormal corpulence always develops, while the growth lengthwise is considerably retarded. (cf. Figs. 229 and 230.) Metabolism is retarded, the interchange of gases lowered. Pronounced constipation is the rule. The genital organs and the sexual development regularly remain early-infantile.

All these cretinous changes are developed to a *very high degree* in cases of thyreoaplasia. Nevertheless, the length of life of these creatures is not always, though often, shortened; cases have been observed for instance, in which in spite of the greatest weakness and total dementia, the 37th year was reached.

The *cause* of the disease, according to the investigations of *Bourneville*, *Pineles*, *Knöpflmacher*, *Erdheim* and others, must always be sought in the *congenital total absence of the thyroid gland;* most exact microscopic investigations (*Erdheim*) have never succeeded in establishing the presence of even a microscopic remainder or a trace of the organ. This fact explains the already mentioned constant maximal severity of all phenomena. The fact that the serious phenomena were not present at birth, and begin to develop at the middle or end of the first year, may perhaps be thus interpreted; that during fœtal life, sufficient thyroid gland secretion is imparted by the mother's blood to the child, and that by the milk of the mother these substances are also given to the child. Why then do not all congenital myxedematists die rapidly of their defect? This peculiar fact seems to be explained, thanks to the investigations of *Pineles*, *Erdheim*, and others, in that in the cases of thyreoaplasia the *parathyroid glandules are found as a rule normally developed. Such patients therefore do not suffer from tetany*, the cause of which we must assume to be organic or functional lesions of the parathyroid glands. It is no far cry to assume a certain (already mentioned) vicarious functioning of the para-thyroid glands as cause of the possibility of a relatively long life.

Of the diagnosis and treatment of this form we shall speak at the end of this division.

2. Infantile Myxedema

This disease was separated by *Pineles* from the wealth of cases of sporadic cretinism, to form a second group. In contrast to thyreoaplasis it is a question here not of a congenital anomaly but of a *genuine disease process developing only in middle childhood*. These children, too, are born sound, and also develop normally usually up to the 5th or 8th year. It is a matter of a *genuine myxedema spontaneum*, only that it attacks young children. Correspondingly it is specially frequent in the countries of spontaneous myxedema, England and Belgium, prefers the female sex, and is not found more commonly in the regions noted for goiter and endemic cretinism than in other countries indifferent in this respect. Concerning heredity, the statements of authors vary; specific hereditary predisposition is apparently not constant;

nevertheless, cases of goiter (*Magnus-Levy*) and acromegaly (*Pope* and *Clarke*) have been observed in the ascendants of such patients.

The cause of infantile myxedema seems to be the same as that of adults; in a number of cases infectious diseases, measles, erysipelas and others, are accused ætiologically; in very many cases, as in myxedema adultorum, no known cause can be found; then the beginning is purely spontaneous.

The disease appears between 5 and 13 years and develops gradually. The children who heretofore were physically and mentally of normal development, and had learned well at school, fall behind: gradually there develop the cretinous changes in the face, the pachydermic skin, the swollen lips, loss of hair, permanent infantilism of the genitalia, absence of menstruation; mental development suffers in degrees varying from slight weak-mindedness to complete idiocy. Growth in length is also retarded, the physiological process of ossification (fontanelles, epiphyses) suffers.

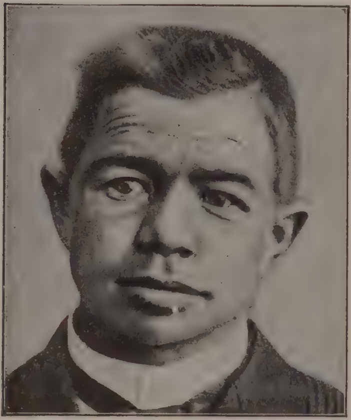


FIG. 231.—Infantile myxedema. Began in the 6th year. At present patient 24 years old (before the treatment). (*From Magnus-Levy.*)

The disturbances in metabolism are equivalent to those of adult myxedema; here, too, there is always anemia, occasionally albuminuria, tendency to hemorrhages, etc.

But we must emphasize that in the cases of this category all physical and mental phenomena of functional loss are usually of a less severe kind. The myxedemoid changes in the skin are often confined to the face alone and not infrequently the patients are capable of some easy vocation. The patient treated by *Magnus-Levy* was, for instance, "moderately developed as to his intelligence, but without defects." In another, the disturbances were so slight that he could be active even as a "legal adviser." Corresponding to the slighter degree of the disease the curative results of organo-therapeutics are often especially good in these cases. All this points with certainty to the supposition that here no congenital defects, but a more or less severe disease of the thyroid gland is the cause of the affection. It has also been assumed with a great degree of probability that in this group the no small number of "thyreoprival" equivalents (*Kocher*) or hypothyriodie benigne chronique (*Hertoghe*) are to be reckoned, among which are also included (as perhaps the most benignant cases) the various kinds of infantilism with sexual aplasia. No certain investigations have yet been made into the *pathologic anatomy* of the thyroid gland in infantile myxedema; the few cases published were rightly placed by *Pineles* in other categories (above all, endemic cretinism). But it is probable that the findings, corresponding to the palpation findings, which were able to establish a very much diminished gland, or none at all, are analogous to those in myxedema adultorum.

3. Endemic Cretinism

This form of thyreoprival disease is probably numerically the most common of all; it has also been known the longest; but to-day its numbers will have to be somewhat decreased, after the exclusion of the other pathogenetic and clinical forms described above. We owe the first fundamental investigations to *Virchow;* later, *Bircher*, *Kocher*, *Curling*, *Lombroso*, *Scholz* and many others threw additional light upon the disease picture.

Endemic cretinism, is as its name indicates, connected with definite regions, appearing in them very frequently: thus above all in Switzerland, Savoy, but also in the Vosges and in the Black Forest. *It occurs frequently only in such regions where goitrous affections are endemic.* In contrast to all other thyreoprival affections, it attacks the *male* sex somewhat more frequently than the female. The progenitors of such patients are often sufferers from goiter; there is often found also a familial and hereditary appearance of this form. *Magnus-Levy* found in the parents of all his patients cretinoid appearance, disturbances in intelligence, etc.

The disease develops probably always in the first year of life, and takes an unusually chronic course. The symptoms consist partly in real disturbances, partly in mere retardation in development. This is always evinced in the growth in height; a dwarf-like stature of 130 to 140 cm. is the rule. At the same time, the osseous system displays rachitic-like changes, bending and distortion of the long hollow bones, a flat and narrow pelvis. Attempts have been made to trace these changes back also to a fœtal rachitis. The abnormal formations of the cranium are especially characteristic; there is usually found a strikingly low and platycephalous type (*Scholz*), a premature synostosis of the cranial bones, and a peculiar shortening of the basis cranii as a result of early tribasilar synostosis (anterior and posterior sphenoid bone and basilar bone) (*Ewald* and others); to this is added the typical change in the osseous and cartilaginous framework of the nose, which in many cases, leads to a veritable "saddle nose."

Besides the dwarf-like stature, the type of face is the main characteristic of the cretin: the forehead is low and receding, the root of the nose drawn in and flat, the nose turned up with nostrils wide open towards the front, the cheek bones protruding in Mongolian fashion, the small slanting eyes slit-like, the lips swollen, the mouth wide, the tongue large and fleshy, the lower jaw broad and strong, but low; as a result of the poorly developed alveolar processes and the deep wrinkles in the skin, there develops a type of aged face. The neck is short and swollen at the sides; the breast is flat, the abdomen protrudes, or hangs like a paunch; umbilical hernias are frequent.

The legs and arms are short, their muscles weak, hands and feet are short and clumsy. Gait is awkward and in some cases seriously disturbed (*Ewald*).

Observations of authors differ concerning the condition of the skin. While *Ewald*, *Bircher* and others found real myxedematoid changes lacking in the skin, and from this fact drew conclusions as to the nosological position of endemic cretinism, *Magnus-Levy*, *v. Wagner* and others have always observed a typical, pachydermic condition of the skin, even though of a lower degree than in the myxedema of adults. It seems, as a matter of fact, to be a question of certain regular, but only gradual differences. The skin of the endemic cretin is also thickened, somewhat gelatinous, but at the same time, flaccid, abnormally movable upon the underlying layer, "has become too wide." The color of the skin is usually pale, yellowish, rarely showing abnormal pigmentation. In lighter cases, but also in more advanced age, the thickening of the skin may become very slight. The same is true also of the increase in the cushions of fat, which are more frequently lacking in endemic cretinism than in other forms of myxedema.

The mucous membrane of the mouth and pharynx frequently shows changes analogous to those in the outer skin. The hair is usually sparse, discolored and stringy; the bodily hair, especially of the genitalia, is nearly always lacking. The changes in the teeth and nails are said to be in many cases comparatively unimportant.

In respect to the internal organs of the thoracic and abdominal cavities nothing is to be specially remarked. Almost constant, on the other hand, is *hypoplasia of the genitalia;* they remain totally undeveloped at an early infantile stage; in female patients there is no menstruation. In lighter cases, however, there is sometimes a late maturity combined with procreative power; the menstrual period may occasionally not appear until the patient is over twenty. In many cases there is anæmia, in most, obstinate constipation. In respect to metabolism, the condition is much the same as that established in the other forms.

The condition of the *thyroid gland* in endemic cretinism is different from that in the other forms of the disease already discussed. While in the myxedema of adults and of children and in thyreoaplasia, there is always present a high degree of atrophy, or total absence of the gland, there is found here in 60% (or more) of the cases, *goitrous degeneration* of the same; atrophy and (palpable) absence of goiter are far rarer (according to v. Wagner only in 10% of the cases); the persons without goiters are from reasons easily understood supposed to be always the most severely affected.

Of the changes in the *nervous system*, the psychic defects stand in the foreground. Often, they consist of total idiocy, occasionally in a mental degeneration lying below the level of brutes; but slighter mental defects also are not infrequent, which occasionally permit occupation with house work (spinning, etc.). In severe cases, speech is reduced to a minimum, restricted to brutish emotional noises; in patients slightly affected, there is a slight disturbance in the power of speech. Deafmutism and difficulty in hearing are very common (*Scholz*). Occasionally, also, diminution of taste and smell have been established. Anatomic investigations of the brain (*Scholz* and *Zingerle*) showed, in part, a simple inhibition in development, in part, inflammatory-degenerative processes.

The **pathogenesis** of endemic cretinism is obscure. At any rate it is rooted in the endemic disadvantages, which region (water supply), climate, hereditary, and other factors signify also for goitrous diseases. While one formerly identified ætiologically endemic and sporadic cretinism, we must now, with *Ewald*, *Pineles*, *v. Wagner*, and most other authors, adopt the point of view that *endemic cretinism is pathogenetically quite different from thyreo-aplasia and infantile myxedema:* nowhere in endemic cretinism has the total disappearance of an already developed gland or aplasis of the same been proved present anatomically (*Pineles*); nay, rather, it is a question of a (probably infectious, not yet clearly known) injury of the *developed organ.* Now, whether hypothyroidism alone explains all symptoms of the disease, or whether with *Ewald* and *Bircher* we must assume still other unknown factors in its origin, must remain undecided, though the first supposition is the more probable.

The **differential diagnosis** of infantile myxedema from endemic cretinism may be deduced from the statements already made. The developed forms of the disease can scarcely fail to be recognized. Nevertheless confusions are possible, especially with simple idiocy and imbecility, which, however, lack the changes in the skin, bones and thyroid gland. Naturally there are gradual transitions between the *clinical* picture of cretinism and idiocy. The same is true of certain severe cases of rachitis with anæmia and scrofula, which may sometimes superficially resemble myxedema, and especially of the typical dwarf growth (nanismus), which occasionally (especially in older dwarfs) produces that peculiar mixture of infantile and aged characteristics, which may also be peculiar to lighter cretinism. Other uncommonly rare diseases in the development of the bones, for instance, achondroplasia (*Parrot*) are differentiated from cretinism in spite of the dwarfed growth, the corpulence and plumpness, by defects of high degree (not mere retardation) in the growth of the skeleton, normal skin and equally normal psyche. Even the mongolism much described recently, lacks the characteristic skin and thyroid gland changes of the thyreoprival disease (cf. the chapter by *Ibrahim*) even though mixed forms (mongoloid cretinism) were observed.

It has already been mentioned that not a few cases of sexual infantilism with corpulence were to be regarded as formes frustes of a hypothyreoidism; these cases therefore can not diagnostically be sharply separated from the cretinoid diseases.

The **treatment** of **infantile thyreoprival diseases** has a varying success according to the form of the disease.

Doubtless the treatment with thyroid gland preparations (cf. above) has the most favorable results in infantile myxedema. Here according to the experiences of *Northrup*, *Ewald*, *Magnus-Levy* and others, even after a few weeks, one can see surprising results (using from 0.18 to 0.3 pro die); children begin to grow, the skin becomes firmer, the cushions of fat decrease, the hair becomes more abundant, the gait improves, and above all the psyche develops, so that children who could up to this time scarcely mumble, quickly learn to talk.

Such marvelous permanent success was displayed by a 14-year-old patient of *Magnus-Levy*, who after a brief treatment, became psychically completely normal, physically completely lost the signs of myxedema, and in three and a half years grew 22 cm. in height. The sexual development also becomes normal in such cases. The patients as a matter of fact become capable of working.

The results of the treatment in the cases of thyreoaplasia seem somewhat uncertain. Nevertheless here, too, quick improvements have been reported (*Magnus-Levy*). Whether complete recoveries occur is uncertain. At any rate, however, *considerable improvements* in psychic and somatic respects are the rule. The way in which the improvement is manifested is analogous to that in aplasia of the thyroid gland.

The therapeutic results in endemic cretinism are different. In severe cases with total or considerable idiocy, one is often unable, according to the experiments of *Scholz*, *Lombroso* and others, to effect cures; indeed, deteriorations have been observed, even cases of death. But on the other hand stand the successes of *Magnus-Levy* and *v. Wagner*, who, however, treated apparently slighter, not fully developed cases. These authors effected improvements in physical and mental conditions, quite equal to those secured in sporadic cretinism.

In any case, the result of all these experiences is *that in every form of infantile myxedema and cretinism, thyroid gland treatment* (*always careful*) *is absolutely indicated.*

Whether the implantation of thyroid glands—especially plausible in infantile myxedema—according to *Kocher* or *v. Payr* methods will be successful, the future must teach us; but it seems very probable indeed.

4. ACROMEGALY

The disease was comprehensively described by *Pierre Marie* in 1886 and supplied with its characteristic name; long before that time, *Verga*, *Friedreich*, *Langer* and many others had described similar cases.

The disease is characterized by *a gradually appearing extraordinary* *increase in the size of certain distal parts, regularly of the nose, chin, thorax, hands and feet; the soft parts and the bones are affected with varying degree of severity. At the same time, there is always found a disease of the hypophysis cerebri, which causes the cerebral symptoms in the malady.*

Symptomatology and Course.—Usually in the third and fourth decades of life, less frequently even in the second, there develop quite gradually, at first unnoticed by the patient, thickenings and (more rarely) increase in the length of the hands and feet, usually somewhat later of the nose, lower jaw, supra-orbital arch, the region of the malar bone and the tongue. These objective changes are usually introduced by "general nervous" symptoms, pressure and pain in the head, lassitude, depression, apathy; often, too, there are found peripheral symptoms, especially of the vasomotor type (acroparæsthesias, asphyxia of the fingers, etc.). Occasionally, however, in spite of the persistence for years of an acromegaly of high degree, all subjective symptoms are lacking so that the patients remain perfectly capable of work up to the acme of the disease. The genital functions suffer very early in a striking number of cases: in men, sexual power is lost, in women, menstruation disappears. Further on in the course, together with considerable increase in the hypertrophic changes there appear disturbances in visual power, and other pressure paralyses of cerebral nerves; synchronously the anæmia, fatigue, physical and psychical asthenia increase. The height of the disease, usually attained after a duration of one or two decades, shows the following *changes:*

The general impression is that of a languid, awkward giant. Often—by no means always—the patients are of abnormal height especially when the disease began during the years of growth. The features are strikingly coarsened, the region of the supraorbital arch protrudes abnormally, while the forehead is receding; under the eyes there are swollen sacks. The nose is heavy and knobby, also somewhat lengthened; the region of the malar bone is prominent. While the upper lip usually remains normal, the chin is considerably increased in volume; the lower lip protrudes, owing to the prognathism of the lower jaw; the ears are large and thick. The cranium is usually not very much enlarged only the tabular portion and the occipital bone are hypertrophied. In the trunk, enlargement of the thorax in all directions especially in depth is striking. Especially noticeable are the considerably enlarged sternum and the powerful collarbones. The upper dorsal part of the spinal column always becomes kyphotic.

But the changes in the hands and feet are peculiarly striking; the hands become soft, spongy, usually pallid paws; often only breadth and thickness are increased, more rarely, .the length. At the same time, the nails (in contrast to drumstick fingers) remain of normal size and flatness, appearing, therefore, comparatively small; usually they are free from trophic changes. There are analogous alterations in the feet. The extraordinary size of the hands and feet, moreover, is all the more striking, because of the fact that arms and legs remain normal, and in later stages, owing to muscular wasting, may appear rather thin.

The *skin* is, in general, thickened, forming great folds and wrinkles; usually it is soft, flaccid, lustreless, moist, but often too remarkably dry. Important trophic or—permanent—vasomotor changes are mostly lacking. Frequently the hair falls out. In the first years of the disease, the cushion of fat is considerably increased.

Of the *internal organs*, macroglossia appears in the digestive tract; the mucous membrane of the pharynx and the œsophagus are occasionally found thickened. The heart also is said to have been enlarged in some cases and the peripheral vessels thickened. Quite rarely hyperplasia of the parenchymatous organs has been established also. As to the condition of the thyroid gland, and the thymus, findings vary; occasionally they were found enlarged, or at any rate persistent; there seem to be no constant changes in them. Very frequently the larynx is abnormally enlarged, the voice rough and very deep.

The genitalia are not rarely hypoplastic; and even pronounced infantilism has been found in not a few cases of acromegaly. (*Uthoff*, *Cushing.*) It has already been mentioned that the function in apparently normal genital organs suffers very early.

The *nervous system* and the *bones* always show considerable changes.

A *change in the hypophysis* has been established above all as a constant finding in acromegaly, either a pure hypertrophy, adenomatosis, partly with colloidal degeneration, or other conditions of degeneration, but probably with most comparative frequency, tumors of the type of the glioma and sarcoma. These tumors which may reach and even exceed the size of walnuts, slowly sap away the bone, destroy, as can always be seen in the X-ray picture, the sella turcica and the sphenoid bone.

The subjective general phenomena caused by the tumor, are the typical symptoms of tumor cerebri: vertigo, vomiting, violent headache, visual disturbances, deterioration of the mental faculties. The most important objective changes are those caused by the hypophysial tumor through pressure on the optic nerve, especially on the chiasma. Ophthalmoscopically (and anatomically) there are mostly found partial atrophies (rarely choked disc) of the optic nerves. Most characteristic is the *bitemporal contraction of the visual field*, easily explained by pressure on the chiasma, which was first described by *Schultze* and naturally is often not altogether symmetrical; occasionally also there are found concentric contraction or even a normal perimetric field. Less frequently, the oculomotor and trigeminal nerves suffer through pressure. Disturbances of the visual function that result from all these lesions are remarkably varied and capable of improvement.

In the cerebral nerves as well as in the peripheral, simple hyperplasia and thickening have been found. Peripheral paralyses occur only in isolated cases, and sensory (neuritic) disturbances, except for pains of various sorts, are extremely rare. The tendon reflexes have been found increased, occasionally decreased, and even abolished.

Of the changes in the *osseous system*, in the cranium, besides the destruction of the sella turcica are found almost constantly, the following: irregular thickenings of the skull-cap, widening of the frontal sinus, rarely of the antrum of Highmore, enlargement of the horizontal rami of the lower jaw, as a result of its prognathism (*Strümpell*), thickening of the tabular portion of the occipital bone. The spinal column shows the already mentioned cervical and dorsal kyphosis with thickening of the processi spinosi (*Sternberg*). In the thorax the mighty collar bones supplied with exostoses are prominent, furthermore, the thickened sternum with the ensiform process, which is usually extremely enlarged. The entire thorax becomes abnormally deep and broad; the ribs also show hypertrophic changes. In the arms and legs only the distal portions (especially radius and ulna) are described as thickened, swollen, "bulbous." Also the bones of the fingers and toes, especially the phalanges are described as thickened and compact, even though their thickening is comparatively far less than that of the soft parts. Frequently there are found in them exostoses of various forms, the ligaments are especially thickened, the vascular furrows abnormally deep. On the whole, therefore, the changes in the bones are hypertrophic. But there are also found, apparently only in the later stages—retrogressive processes in the bones; the favorite places of these atrophies which are often of high degree, are almost invariably the distal parts of the ulna and the phalanges of the toes (*my own observation*).

Of disturbances in the *organs of digestion*, nothing particular need be mentioned except the rarely described polyphagia and obstinate constipation. More important are the changes in *metabolism*, especially the common *diabetes mellitus*, less frequently a diabetes insipidus. The former may display the typical signs of "severe" diabetes and cause death through coma. Whether this diabetes is pancreogenous, or—what is more probable—is caused by pressure on the fourth ventricle, has not yet been decided.

Concerning the condition of oxidation, it is found by many authors to be increased (*Magnus-Levy*); respiration experiments of other authors (*Salomon*, *Schiff*) showed a normal condition. The *various stages* of the disease are sufficient explanation for the variety of these findings.

Epilepsy, paralysis agitans (*Bruns*), myxedema, Basedow's disease, dementia myoclonica (*Oppenheim*) have been observed as rare *complications* of acromegaly.

Prognosis.—The course is usually extremely slow and insidious; mostly it extends over several decades (up to 50 years). Less frequently an acute malignant course in 3 to 4 years was observed (*Sternberg*). Occasionally, there are sudden apoplectiform deteriorations, with acute blindness, paralyses of the cerebral nerves, for instance, of the oculomotor (*Sternberg*, author). But these acute disturbances may, in many cases, be cured. Long remissions without demonstrable local or general deteriorations are common. *Tamburini* and later *Hutchison* assumed two anatomically and clinically distinct stages: the hyperplastic stage and the cachectic stage with retrogression of the hyperplasias, a division for which the observation of numerous clinical cases, the appearance also of the already mentioned *retrogressive* changes in the bones seem to speak. The death of the patients occurs either in diabetic coma, or not rarely suddenly under signs of cerebral paralysis; often, too, various complications (tuberculosis, heart trouble, etc.) put an end to life.

The **prognosis,** therefore, is nearly always unfavorable quoad sanationem.

Internal **treatment** has so far been powerless. The supposedly observed improvements, induced by organo-therapeutic preparations (thymus, hypophysis tablets) are probably to be interpreted rather as spontaneous remissions. Occasionally, I have seen pronounced deteriorations resulting from hypophysis treatment. Whether surgical treatment, extirpation of the hypophysial growth (*Calon* and *Paul*, *v. Eiselsberg*, *Schloffer*) which has great technical difficulties can attain permanent results, the future alone can show, but it seems probable, judging from the recent successes of operative treatment.

The **pathogenesis** of acromegaly is still obscure, in spite of the painstaking study numerous authors have devoted to it. Opinions, especially as to the rôle of the hypophysial tumor, vary considerably, since the physiological function of the gland is still unknown. In the discussion of this question we meet a striking analogy to the thyreogenous theory of *Basedow's disease*. Experimental investigations point to a functional connection with the thyroid gland (hypertrophy of the hypophysis after thyreoidectomy). The hypophysis is supposed to extract certain substances from the blood and to return them again after alteration, that is, to work antitoxically. But the extirpation of the gland in animals has—so far—never led to trophic disturbances like those of acromegaly, but only to nervous symptoms and cachexia.

Some authors regard the *increase* in the functioning of the gland as the cause of acromegaly (*Tamburini*, *Benda*, and others), others think it due to a *change* of secretion, a dysfunction. *Strümpell*, *Arnold*, and others, on the other hand, consider the hypertrophy of the hypophysis only a co-ordinated and not the causal phenomenon. Against the theory of hyperfunction speaks the fact that not rarely, malignant, *function destroying* tumors have been found in acromegaly. In such cases, the course of the disease is abnormally rapid. Against the hypothesis of only co-ordinated hypertrophy speaks the same fact, that actually, in many cases, the gland was not merely hypertrophic, but also sarcomatous and gliomatous. That the *loss* or diminution of function *alone* can not produce acromegaly is proved on the one hand, by the negative results of animal experimentation, on the other, by the numerous cases, in which hypophysial tumors (usually of malignant character) were observed without acromegaly, or equivalent trophic disturbances (*Kollaritz*, *Berger*, and others). Finally, we must mention the hypothesis that not the hypophysial tumor itself, but its irritating (pressure) influence upon definite, assumedly trophic centers of the brain may be the cause of acromegaly (*Erdheim*).

From all this, only one thing seems clear, that the somatic or functional change in the hypophysis has in it a component for the occurrence of acromegaly. It is still quite obscure what other disturbances must be added to evoke the disease.

The **differential diagnosis** of the *developed* disease is always easy, especially when the findings in the eyes and the X-ray picture prove the presence of a hypophysial tumor. In such cases, certain forms of syringomyelia with pronounced enlargement of hands (macrocheiria), many cases of myxedema, of lymphatic habitus, of adipositas of varying forms (adipositas dolorosa, lipomatosis perimuscularis circumscripta, *Curschmann*), of familial (yet physiological) abnormal size of hands and feet and other such diseases, could scarcely cause differential diagnostic difficulties.

In respect to differential diagnosis, furthermore, *general and partial gigantic growth* must above all be considered, the former especially because, on the one hand, 40% of all known "giants" suffer from acromegaly, and, on the other hand, a large part of patients with acromegaly are giants (*Sternberg*). The absence of all pathologic acromegalic symptoms, especially of a cerebral hypophysogenous kind in the "normal giant" makes the differentiation easy. Partial giantism (usually congenital, very rarely acquired) attacks usually only single extremities, bringing about the most remarkable deformations, or one half of the face, or occasionally too one half of the body, and scarcely ever appears symmetrically; therefore this anomaly is nearly always easily differentiated from acromegaly.

In differential diagnosis from incipient acromegaly, the following may furthermore cause difficulty: the vasomotor-trophic neuroses with swelling (for instance, angioneurotic edema of the hands, erythromelalgia), chronic inflammatory conditions of the skin, tendon sheaths and joints (leucæmia cutis, and leprosy, which may both be associated with facies leonina), many forms of deforming arthritis and incipient elephantiasis. Finally, are especially important, a number of diseases of the bones with hypertrophy and deformation. *Osteoarthropathie hypertrophiante* (*P. Marie*), which leads to enormous knob-like swelling of the finger tips with pronounced convex deformation of the nails (in contrast to acromegaly); the "drumstick-fingers" of some sufferers from heart and lung trouble are reckoned here, not always justly, since in them the bones are completely normal (*Dennig*). Osteitis deformans (*Paget*), diffuse hyperostosis (general or confined to the skull) described by *Virchow*, as leontiasis ossea, finally also the cranium progeneum (*L. Meyer*), which appears not only in sufferers from acromegaly, but also in cretins, psychopathics and degenerates, and in rare diseases of the bones, may have a greater or less similarity to acromegalic changes, but always lack the classical nervous and hypophysogenous phenomena. In myxedema, the differential diagnosis is supposed to have difficulties because, here too—though rarely—hypophysis changes, even bitemporal hemianopsia, have been observed. But usually these hypophysis enlargements in myxedema attain only the slight volume of a vicarious enlargement, as which they should be interpreted, quite analogous to the enlargement and persistence of the thymus gland in similar goiterous degenerations.

Finally we must indicate a peculiar kind of acromegaly which has been recently observed in rare cases (*Fröhlich*): cases of general abnormal *lipomatosis*, with aplasia or functional disturbance of the genitalia, bitemporal hemianopsia, but without any special change in the distal portions of the body. This disease picture of which I also was able to observe a case, is unquestionably likewise evoked by a disease of the hypophysis. Recently affections of the *pineal body*, too, were reported to be the cause of such an adipositas (*Marburg*).

5. HEMIATROPHIA FACIALIS PROGRESSIVA

(*Neurotic Facial Atrophy, Romberg's Disease*)

This very rare disease observed first by *Parry* (1837), later classically described by *Romberg* the elder, attacks usually young females, much less frequently males in the second and third decades of life; very seldom does it occur after the 30th year. The onset is usually slow, insidious; very frequently prodromes precede for many years in the form of neuralgic symptoms such as "toothache." Usually the disease breaks out soon after a traumatism, the extraction of a tooth, an otitis media, diphtheria, pneumonia and other infectious diseases. It begins nearly always in a relatively small spot in the cheek over the malar bone, in front of the ear, and in other places; often this spot is described as infiltrated or "knotty." Gradually the skin of the face on the side affected becomes atrophic, glossy, thin as paper, often abnormally shining, still more frequently (as in many atrophies of the skin) discolored to a light brown. The atrophy of the skin spreads usually irregularly in the form of patches and stripes; in the latter case arise the linear indentations which French authors call "coups de sabre."

Simultaneously—often, too, considerably later—the soft parts and the bony framework of the face become atrophied; the fatty cushion of the cheek disappears, the cheek becomes hollow; the eye sinks farther in than is normal; especially prominent is the diminished but still protruding malar bone, because of the atrophy of the soft parts, and the narrowing and shortening of the affected side of the nose. The side of the forehead that is attacked recedes. The contour of the lower half of the face becomes very asymmetrical, especially because of the atrophy of the mandibula. The atrophic process always ends precisely in the middle line of the face. Here there is frequently found as boundary a slightly prominent pigmented stripe. Occasionally the tongue is atrophied unilaterally; very frequently also the soft palate, the larynx, the vocal cords, the concha of the ear, probably never the eyeball. The muscles of the facial region, and the chewing muscles mostly seem somewhat atrophic, but are usually only little changed in function or in their mechanical or electrical condition (and always only quantitatively). In some cases, tonic and clonic spasms of these muscles have been observed. The hair on the side affected (usually only eyebrows, eyelashes and beard, very rarely the hair of the head) falls out or becomes colorless or often white.

Changes to be interpreted as disturbances of the function of the sympathetic are comparatively rare; they consist in unilateral vasomotor disturbances, paroxysmal or permanent pallor and coolness, occasionally in the loss, less frequently in the increase of the secretion of the sebaceous and sweat glands; in some cases *Horner's* oculo-pupillary symptom complex (contraction of the palpebral fissure and the pupil) has been observed, in others tachycardia.

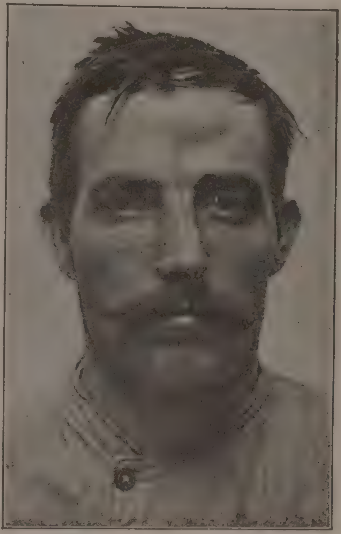


FIG. 232.—Hemiatrophia facialis sinistra especially of the forehead. (*From Schoenborn-Krieger.*)

Of subjective symptoms, the violent neuralgic pains of the trigeminal region, which appear as prodromes as well as during the completely developed disease, stand quite in the foreground; often also there are general or unilateral headaches, always naturally, a distinct sensation of tension on the diseased side. Real disturbances in sensibility (hypæsthesias, anæsthesias) are very rare. The lower jaw reflex is—in so far as it has been tested—described as normal. Disturbances of the sensory cerebral nerves (besides the fifth) are mostly lacking.

Rare Forms.—Sometimes an extension of the atrophic process to the shoulder on the same side, to the upper arm, one breast, even to an entire half of the body has been observed. In a few cases, there was added to the hemi-atrophy an atrophy of the other half, so that a total facial atrophy, just as in extreme scleroderma resulted. In extremely rare cases, a paralysis of some cerebral nerves, facialis, acusticus, recurrens, etc., upon the same side has been observed (*Gowers*), which arose from compression in the atrophically narrowed(?) foramina of the cranium. Complications of the disease with other nervous diseases arising on a degenerative basis has been observed none too rarely, as with chorea, cramps, spasms of the musculature of the face, tabes, multiple sclerosis, scleroderma, etc.

Course and Prognosis.—The course is always exceedingly chronic and extends over many years. Finally, there is a standstill in the process. Life is never endangered by the disease. But the prognosis quoad sanationem is nearly always unfavorable. Improvement was observed very rarely; the possibility of retrogression of existing atrophic changes is excluded.

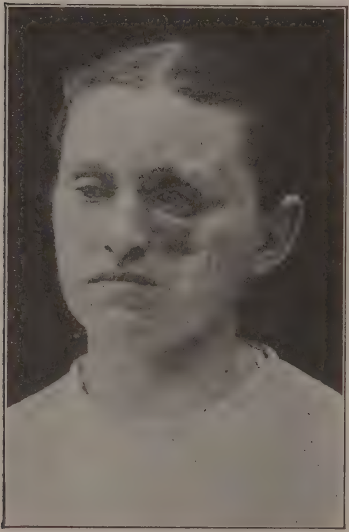


Fig. 233.—Advanced case of hemiatrophia facialis sinistra. (*From Schoenborn-Krieger.*)

Treatment.—The galvanic current may, according to the experience of *J. Hoffmann*, always be tried, although in the majority of cases, it had no effect. In one case, *Oppenheim* saw an improvement following the resection of the cervical sympathetic. Considering the rarity of disturbances of the cervical sympathetic in hemiatrophy, it is not probable that this treatment has any future. By some authors injections of paraffin to compensate the defects were employed with good cosmetic results. Internal remedies have always failed.

The **pathogenesis** of hemiatrophy—one of the most interesting questions—is still decidedly obscure. Opinions of authors have always been diametrically opposed. *Bergson* and others assumed an alteration in the vascular nerves, a transitory or permanent local vasomotor constriction as the cause. *Romberg* declared the disease to be a pure primary trophoneurosis. *Samuel* assumed on the basis of Romberg's view, a lesion of the centripetal and centrifugal trophic nerve tracts; he saw in the unilateral facial wasting precisely the proof of the reality of specific trophic nerves, as to the presence of which there is still much discussion, though it is mostly denied. *Virchow*, *Mendel*, and others, declared that affections of the trigeminal nerve, either of the trunk or of the centrally situated parts, were the cause of the trophic disturbance, without however being able to explain the contradiction inherent in the extraordinary intactness of the sensibility and the chewing muscles. Möbius was directly opposed to this trigeminal hypothesis. He assumed the effect of a local poison of such a kind that "a poison enters through the skin or mucous membrane, perhaps connected with bacteria and that this poison slowly increasing, causes the skin to waste." It is evident enough that this theory explains neither the strictly unilateral character of the affection, nor certain very frequent often prodromal trigeminal symptoms (for instance the neuralgias).

Other authors (*Seeligmüller*, *Oppenheim*) declared disturbances of the cervical sympathetic to be the ætiological factor. This explanation seems but little plausible if one considers how comparatively rare in the disease are typical cervical sympathetic disturbances on the one hand (*F. Lange* found them described only 18 times in 163 cases in the literature) and on the other hand how very, very frequently unilateral sympathetic disturbances which run their course without any trophic changes have been observed.

Jendrassik finally sees the seat of the disease neither in the fifth nerve, nor in the cervical sympathetic; rather he suspects it to be in a place where parts of the sympathetic and trigeminal are close together, that is, at the base of the skull; here, the carotid plexus and the ganglion Gasseri are close neighbors. *Jendrassik* assumes a lesion of the sympathetic cerebral ganglia or of the Remak fibres connected with them, as being of ætiological importance.

The trigeminal hypothesis, in spite of the opposition of *Möbius*, *Oppenheim* and others, seems to me really worth discussion. For there are unquestionably certain nerves (above all I mention the n. medianus) which react to lesions of any sort preponderantly by *trophic* disturbances, before which, the other phenomena of functional loss, both motor and sensory, may step into the background. From analogy with the n. medianus, it seems to me quite plausible that there may be certain disturbances which, as a rule, injure only the trophic functions of the trigeminal, more frequently causing certain sensory phenomena (neuralgias), (without leading, as a rule, to hypæsthesias) and cause at the same time no *motor* phenomena of functional loss. As a matter of fact, attempts have been made—since most patients are young—to assume as the predisposing factor a congenital weakness of the hypothetical trophic centers for the side of the face which is attacked.

The **differential diagnosis** of the *developed* disease needs no special discussion, since the picture of the totally developed hemiatrophia facialis is exceedingly characteristic; at most, we may say that unilaterally localized scarred surfaces from burns or ulcerations (lupus, etc.) may at first sight remind one of hemiatrophy.

The differential diagnosis of incipient facial wasting may be more difficult. Here we must mention before all *scleroderma* which occasionally, beginning en plaques, may attack the face also; but this localization of scleroderma is extremely rare. Usually it is distinguished from facial wasting by appearing in pronounced symmetry, furthermore, by the fact that it lends the atrophic skin a peculiar hardness and that in the beginning it spares the bones. Finally, the sclerodermic hands and fingers which are nearly always affected together with the face, insure differentiation of this disease from hemiatrophy.

Bone atrophies after traumatism (*Sudek*), which progressing very slowly, may evoke circumscribed changes in the shape of the face, are also concerned in differential diagnosis, since, as I observed, the skin over such atrophying bones (for instance on the forehead) may be somewhat thin and pigmented, but this bone atrophy is probably always easily distinguished from hemiatrophy by the fact that it remains somewhat localized and never attacks an entire half of the face; here also, the atrophy of the *bone* stands fully in the foreground (in contrast to *incipient* hemiatrophia facialis).

Finally, we shall have to mention certain diseases which may leave asymmetries of the face, even of the entire head as residues of anomalies of growth, or as anomalies in growth dependent on mechanical factors. In cerebral infantile paralysis (especially porencephalia) there is occasionally found a striking retardation in the growth of the paretic half of the face. Considerable asymmetries of the halves of the face are also observed in torticollis congenitus. The persistently normal condition of the skin and the absence of all progress in the atrophy naturally facilitate differential diagnosis from facial wasting. The same is true of asymmetries caused by an early unilateral total loss of teeth, as I observed once in syringomyelia.

It has been already mentioned in the chapter on acromegaly that in extremely rare cases, there occurs also a *hemi-hypertrophia faciei.*

XI

INTOXICATION DISEASES OF THE NERVOUS SYSTEM

BY

F. QUENSEL (Leipsic)

1. POISONING BY THE CARBON COMPOUNDS OF THE ALIPHATIC SERIES

(a) Alcoholism

Judged by prevalence, alcoholism represents the most important of all the intoxications with which neuropathology is concerned. Its real cause is the absorption of the ethyl alcohol (C_2H_5OH) that is present in all spirituous liquors, 3–5% in beer, 5–20% in wine, 25–50% in brandy, while all the other constituents, fusel and ethereal oils, esters, acids are only secondarily concerned.

As in all toxic diseases, besides the poison, individual *susceptibility* plays an important part, especially in the causation of chronic diseases. Men with an *inborn*, usually an *inherited* predisposition, succumb especially easily to alcoholism, a predisposition which may be discovered from the previous life of the patient, from the occurrence of nervous and mental diseases in the progenitors, etc. Especially endangered are the progeny of drunkards. Often there is found an *acquired* disposition as a result of preceding injuries to the head, of other poisoning, of syphilis and other infectious diseases, typhoid, malaria, etc. Frequently this susceptibility is shown early by an *intolerance* to alcoholic liquors, which even in really small quantities, is followed by disturbances.

As to the essential effects of alcohol upon the nervous system, in isolated or brief absorption, both experimental tests and the conditions of simple intoxication inform us that even small quantities may evoke pronounced changes, especially of the psychic functions, decrease in the clearness of sensory perception, impairment in thought and judgment, dulling of the finer emotions, at first facilitated excitation of movements, later inhibition and disturbances in the coordination of movements. In higher degrees of alcoholic influence, there is a loss of all inhibitive influences, dimming of consciousness, finally, motor and sensory paralysis. This is the picture of acute alcoholic influence, which, however, varies with the individual.

Such an isolated intoxication, even one that is accompanied by lighter symptoms, leaves behind it certain permanent effects, from simple diminution of the physical and mental capacities, to pronounced somatic and psychic disturbances. The most important after-effect is that the use of alcohol always leaves behind a certain hunger for new absorption of the poison, and that after repeated drinking, the habit develops as a passion for alcoholic liquors. From this individually different, suddenly appearing moment, one may speak of *intoxication*, and its result, chronic alcoholism.

Symptomatology.—To the phenomena of *chronic alcoholism* belong also the results of other organic changes in the body, changes in the liver, kidneys, heart, vessels, blood, etc., a bloated reddened face, venectasias, acne rosacea, dull glance, swimming eyes, catarrhal mucous affections of the larynx and pharynx, the odor of alcohol on the breath, catarrh of the stomach, morning vomiting, etc. In the earlier stages the patients are often enormously corpulent; later they present, in general, a prematurely aged and dissipated appearance, the characteristic picture of the degraded brandy fiend.

The most striking of the somatic nervous phenomena, is *alcoholic tremor*, a trembling with fine and rapid oscillations (8–12 oscillations per second), which appears especially when the fingers are spread, and in all movements. If one places the tips of the patient's extended fingers perpendicularly on the palm of one's hand, one feels a peculiar grating (Quinquaud's phenomenon). One often feels the trembling very clearly even when it is seen only with difficulty. The finest fascicular-like muscular twitchings are often found in the tongue, the lips, and the other musculature supplied by the facial.

Severe motor disturbances do not belong to the picture of simple alcoholism, but frequently there is a considerable flaccidity of the musculature, especially in the lower extremities. Painful cramps in the calves of the legs are common.

Sensibility to touch is occasionally slightly dulled, but that for pain rather increased. Manifold visual disturbances of various kinds are found, decrease in clearness of vision, nyktalopia, concentric narrowing of the visual field, or central scotoma. Painful *pressure points* are frequently found in the most varied places, and not only are sensitive in the typical places, the nerve trunks, but often the musculature especially, that on the anterior aspect of the thigh or preferably on the calves. Not rare are spontaneous pains, appearing paroxysmally, neuralgia-like, as sciatica or lumbago, or even more permanently, pains in the nape and temples, etc.

The tendon reflexes in uncomplicated cases are not changed; they may be comparatively weak, or, more rarely somewhat increased; the cutaneous reflexes are usually very active. In simple alcoholism, the pupils show no characteristic disturbances; still one often finds a comparatively slow light reaction approximating the pronounced slow reaction that occurs in the pathological conditions of intoxication. Of secretory disturbances, there is mainly an inclination to profuse outbreaks of sweat, while excessive secretion of saliva usually depends upon the presence of catarrh of the larynx.

In chronic alcoholism, the *psyche* undergoes a gradually deeper and deeper change. The mental capacity, on the whole, decreases, interest diminishes, there is a decrease in the power to receive and retain new impressions, and in memory; the total absence of insight into his own condition is but rarely and temporarily relieved by the repentance of the "Katzenjammer" ("the blues" following intoxication). Dulling of the feelings, brutalization, irritability often lead to deeds of violence and to criminal passion, and the end is frequently a greater or smaller degree of dementia. The course, however, is by no means always so progressive, and may halt at any stage.

Upon this foundation, there now appears a series of circumscribed nervous and psychic diseases.

Frequently the general nervous disturbances and subjective troubles assume a specialized form, corresponding to the picture of the known neuroses. Pain and pressure in the head, muscular weakness, fatigue, sleeplessness, circulatory and digestive disturbances, increased sexual excitability or impotence, etc., present the picture of an *alcoholic neurasthenia*. In other cases, the psychic condition, lability of the emotions, suggestibility, etc., has a marked appearance of *hysteria* (cf. the chapters concerned) especially the somatic phenomena, anæsthesias, hyperæsthesias, phenomena of paralysis, contractures, etc., are found upon an alcoholic basis; on the other hand, the susceptibility to psychic influence is not always very pronounced in alcoholics. It is worthy of mention that in addition to the usual alcoholic traits, often are present certain light organic changes—and this holds good for many other toxic diseases likewise—a sign of the deeper basis of the disease. As it seems, these symptom-complexes appear earlier in individuals already predisposed than in others.

This predisposition plays an especially important rôle, according to manifold experience, in *epilepsy*, this neurosis, that can scarcely be regarded as a functional neurosis, and which may be considered most characteristic of alcoholism. Genuine alcoholic epilepsy has certain peculiarities: first, appearance rather late in life, rare but mostly typical severe attacks, regular relation as to time with other alcoholic diseases, especially delirium tremens. But there also occur frequently attacks of varying form, petit mal, etc., which like genuine epilepsy may lead to dementia. Often, doubtless, alcohol is only an exciting or aggravating factor in genuine epilepsy. It is especially dangerous combined with injuries, concussion of the brain, other poisonings, for instance, with lead, etc. The frequent appearance of genuine epilepsy in the descendants of drunkards is remarkable.

One of the most important nervous diseases that follows very frequently upon alcoholism, is *polyneuritis* (cf. under this heading). We may point out here only some peculiarities—attack by preference of the lower extremities, of the extensors of the lower leg, and the peroneal musculature, symmetrical distribution, hyperalgesia, besides anesthesia, the usual intactness of bladder and pupils. Skin phenomena of vasomotor-trophic character are common while polymyositic symptoms are rare. The involvement of the optic nerve is important, usually in the form of temporal paleness of the discs appearing occasionally, also as genuine papillitis or retrobulbar neuritis.

Combined with polyneuritis, but also alone, that is combined with other phenomena of chronic alcoholism, there is found especially in drinkers of spirits a very severe disease picture, *polioencephalitis hæmorrhagica superior* (Wernicke). In this, there are combined in acute development within about 8–14 days until the end, cerebral general phenomena—vertigo, headache, stupor, nuchal rigidity, deliria—localized cerebral symptoms, disturbances in coordination, gait and speech, but above all characteristic phenomena of paralysis in the realm of the external, often too of the internal muscles of the eye. The outcome is usually fatal.

A comparatively rare disease picture too is *pachymeningitis hæmorrhagica interna*, which by no means need always lead to marked phenomena intra vitam. In some cases, especially with larger hemorrhages from the newly formed vessels, there may appear cerebral sensory and motor phenomena of paralysis, and spasmodic symptoms. Well developed epileptic or even Jacksonian attacks occur often; pressure in the meninges may lead to pronounced choked disc; nuchal rigidity and fever may also be found.

Among the psychic affections of alcoholists the most important and the most characteristic type is *delirium tremens*, of which thousands of persons suffer every year in Germany. Its genesis is not precisely known. An antecedent condition is a long misuse of spirits. Deliria are rare in drinkers of beer alone, and then present a different picture. Occasionally the delirium breaks out in the midst of health; usually prodromes, slightly delirious conditions, sleeplessness, restlessness, etc., precede, sometimes (in about 10%) epileptic attacks. The exciting cause is often found in physical diseases, injuries, fractures, quite frequently gastric troubles, febrile catarrhs, pneumonia, other infectious diseases, etc. The significance of sudden abstinence as cause of the outbreak has not yet been explained; in many cases, it seems to cooperate, but it is probably never the only cause.

The picture of the completely developed delirium is unmistakable—total *disorientation in space and time*, with consciousness of his own personality intact, illusions and *hallucinations* especially in seeing, hearing, and feeling, usually quite numerous small objects as mice in lively motion, little black men, etc., which as in a dream, combine into a peculiarly fantastic delusive situation. In great *motor restlessness*, the patient reacts actively to his sensory delusions, lives in his usual activities (occupational, bar-room delirium), grasps his situation with full understanding. He is hasty, inattentive, easily distracted, easily influenced by suggestion (creation of hallucinations, by encouragement, but also by pressure upon the eyes, or by covering them). The *mood* varies between cheerfulness (drinkers' humor) and anxiety, corresponding to the nature of the sensory delusions, the always transitory insane ideas.

Of physical phenomena, one must mention first the characteristic *tremor* often of coarse oscillations, especially found in the out-spread fingers and the tongue, often visible over the entire body, marked reddening of the skin, *perspiration*. Often there are light pareses in the facialis, in the tongue, disturbances in coordination, weakness, stumbling over syllables, which are conditioned only in part by psychic distraction, in part also by concomitant neuritides.

Frequently there occurs (whether without any concomitant organic affections, catarrh of the stomach, bronchitis, etc., is uncertain), a more or less high fever; the *frequency of the pulse* is heightened even in rest, as are also respiration and the pressure of the blood. In the urine, one mostly finds *albumen*, often too hyaline casts, not rarely acetic acid, and some sugar. Likewise a change is observed in the composition of the blood, decrease of the eosinophilic, increase of the usual neutrophilic leucocytes.

All these phenomena rapidly attain a considerable degree and last on an average from 4 to 7 days. There are also more protracted forms, and those, which from the duration and development of the symptoms, one must designate as abortive. As a rule, then, there appears recovery in a critical *sleep*, from which it is difficult to arouse the patients. Not infrequently the patients still remain for days under the influence of certain delirious experiences and insane ideas. Rarely is clear memory of the time of delirium retained, but just as rarely is it totally lost.

The outcome in the majority of cases is favorable. The mortality in uncomplicated cases amounts to 1–5%. Death in delirium is more frequent in those cases that are complicated by severe physical diseases, surgical injuries, concussion of the brain, typhoid, etc. Recurrences have frequently been observed (in one patient 27 times) when the abuse of alcohol is continued.

The **diagnosis,** in consideration of the ætiology, is not difficult, but one must consider that occasionally purulent meningitis of the convexity and progressive paralysis of the insane present a similar disease picture, that senile dementia (presbyophrenia of Wernicke) occasionally has a course with similar phenomena. Finally the matter of complications with other nervous diseases (tabes, etc.) must always be investigated.

A rarer acute psychosis closely related to delirium tremens is alcoholic insanity (*acute hallucinosis* of Wernicke), characterized by numerous hallucinations especially in hearing, with retained external orientation, connected with the most intense feeling of terror, whereby there appears the development of a more or less circumscribed logically worked out systematized persecution mania. This hallucinosis occasionally presents transitional forms to genuine deliria, with which it shares the physical phenomena in more or less pronounced form. It is in itself curable, but usually it takes weeks, even months, until the residuary insane ideas finally disappear. Attempts at suicide are frequently made by such patients.

Conditions of genuine depression, upon an alcoholic basis, are very rare.

A subacute or chronic disease, the frequency of which has only been recognized in recent times, is *Korsakoff's psychosis.* Most characteristic of this is the *loss of recent memory.* The patients are unable to retain new impressions, and therefore lose memory of the immediate past, usually from the beginning of the disease. They fill out the gaps with *confabulations;* hallucinations and detached insane ideas are less common. As a rule, especially at the beginning, the patients, as in delirium tremens, are *disoriented* with relation to space and time, retaining however, consciousness of their own personality. There need be no real dementia, but as a rule the patients have lost their critical faculty. The mood is morose or more frequently corresponds to drinker's humor. Because of the frequent though not invariable combination with polyneuritic symptoms, the disease has been called *polyneuritic psychosis.* It often develops from acute alcoholic psychoses, especially from delirium alcoholicum. The disease may be cured, but not infrequently becomes transformed into a permanent dementia. Death results as a rule, only from complications with tuberculosis or syphilis, etc., but is occasionally due simply to marasmus. One must remember that a concussion of the brain and senility may, upon occasion, apparently without cooperation of alcohol, lead to similar disease pictures.

Other chronic psychic disturbances of habitual drinkers develop from the already described, especially psychic, peculiarities of chronic alcoholists. As a result of the impotence, irritability, prejudiced ideas, and conflicts in his family life, arising from his habits, there develops a prognostically unfavorable *jealous mania* of alcoholists. Occasionally there are found some permanent habitual hallucinations, from which temporary, as a rule not very serious, insane ideas develop.

The appearance of dementia, especially with polyneuritic symptoms and the similarity of such disease pictures to taboparesis, has led to the construction of a so-called *alcoholic progressive paralysis*, but the occurrence of the genuine form of this disease upon a purely alcoholic basis, has not been proved.

If susceptibility plays a certain rôle in the origin of all alcoholic disturbances, there are some forms which develop only on the ground of a quite special, pronounced predisposition. Clinicians have spoken of a *pathological condition of intoxication* (complicated intoxication), when in individuals, after the consumption of comparatively small quantities of alcohol, disproportionately severe, peculiar phenomena appear—severe dimming of consciousness with usually slight physical disturbances, great motor restlessness, even to the picture of a furious storming rage, which after a deep sleep in a very short space of time, and usually with complete loss of memory, passes into the normal condition again.

Dipsomania is a form of disease now often credited to epilepsy. The otherwise habitually sober patients begin, as a rule, after an introductory depression, which lasts for days and weeks, to give themselves up in the most unrestrained manner to the enjoyment of alcoholic liquors. Though they are often relatively but little influenced at first, usually they stop drinking only when they are totally intoxicated; after becoming sober again, they are as self controlled and abstinent as ever before, until the next attack. These conditions must be distinguished from pseudodipsomania, in which patients brought upon occasion in quite a normal way, to partake of drink, fall into such a drinking period.

Pathological Anatomy.—As to the anatomical changes in the nervous system in alcoholism, but little is known with certainty. The destruction of ganglion cells in the nervous central apparatus has been experimentally proved. In human pathology, changes are present in all elements of the nervous system. To these belong neuritic processes in the peripheral nerves, in the opticus, in the central nervous system—fibre and cell degenerations, increase in the supporting tissue, changes secondarily evoked by alteration of the vascular system, arterio-sclerosis with atrophy and softening, pachymeningitis hemorrhagica interna, chronic diffuse leptomeningitis, polioencephalitis, hydrocephalus internus. It is certain that some disturbances are only secondarily brought about through changes in metabolism.

Treatment.—The treatment of the individual alcoholic diseases, in so far as they appear quite like or even similar to those of other ætiology, is directed according to the general rules, and may be found in the chapters concerned. The psychoses, likewise, are, on the whole, to be treated according to general psychiatric principles and such patients usually need institutional care.

As a matter of fact, only *delirium tremens*, which is frequently the object of general treatment with all physicians, needs special mention. Because of the many possible complications, because of the danger of suicide and deeds of violence, the patient should be taken, if possible, to a suitable institution, though it must be confessed that the disorientation and restlessness occasionally increase at first after the removal to a new milieu, but this can be met by absolute insistence upon rest in bed, in sufficiently lighted rooms and under constant observation. The possibility and success of this method are evident from the experience of very large institutions. Isolation should be attempted only under continuous observation in suitable rooms. Possibly, lukewarm or shorter cool baths may be used for quieting the patient. From the very first, almost without exception, one may cease the administration of alcohol, it being indicated only when there is danger of collapse. Furthermore, one may confine one's self to giving milder stimulants, strong coffee and camphor (0.1 several times in powders or as injections). Care must be taken that the patients get ample nourishment, especially too, that milk be included in the diet. Soporifics are to be used only occasionally in peculiar external circumstances; one should choose those that have no cardiac effect, veronal 0.5–1.0, paraldehyde 4.0–6.0. In surgical cases, since the patients exhibit marked insensibility to pain, hence pay not the least attention to the most severe injuries, mechanical confinement should be used though otherwise strictly forbidden.

Here, too, as in all similar cases the withdrawal of alcohol is always the most essential part of a rational treatment.

For the alcoholist, the attainment of *total abstinence* is in almost all cases the conditio sine qua non of permanent recovery, and the prevention of more severe relapses. How this is to be brought about, is a question that must be answered according to circumstances. The medicines, chloride of gold, strychnine, which were formerly employed as curative means and to frighten the patient from drinking, have proved to be of no avail. Occasionally, in suitable cases, hypnosis is effective. As a rule, the patient, at first at least, needs institutional treatment, for but few can by their own will power abstain from the poison.

For the higher classes there are open in this case, sanatoriums and institutions for treatment; for the great majority, *institutions for the cure of drunkards* are unquestionably necessary. It is a misfortune that there are really not enough of them; only the distinctly psychopathic alcoholist is suited for insane asylums. Under institutional supervision at any rate, total abstinence can at once be effected. The patient comes among people all of whom abstain, where abstinence is taken as a matter of course, and endures it easily without further protest. In this he is assisted by industrious work, and by a persistent influence in the direction that the drinker himself comes to be a fighter for abstinence and against alcoholism. It is only necessary that his stay should be sufficiently long. This unfortunately causes some difficulties. Entrance is itself hard to procure, and the possibility of disqualification, according to the civil law code, has proved efficient neither here nor for the abstinence. Frequently, moreover, there is not sufficient money for the support of the drinker and his family. Therefore, taking up of the care by the government and public institutions for the cure of alcoholists are to be desired. The prejudices against them must also be combated. A desirable measure and one that has proved successful in many places, has been the erection of institutions (conducted by physicians) where advice and treatment for drinkers may be had.

Furthermore, one must see to it above all, that the patient even after dismissal from the institution, should be kept from the use of alcohol. The duration of the treatment is of great importance here, but still more important is the creation of suitable surroundings, which is frequently attained only by the drinkers joining an abstinence club (Good Templar Lodge, Blue Cross, etc.).

But in order that all these measures be as effective as possible, there is need of a *prophylaxis*, affecting not only the individual drinker, but the entire population. Germany yearly spends three billions of marks on alcohol, a loss that includes also the loss of an enormous amount of the people's health, working power and money. This condition can be changed only if the general attitude towards alcohol changes. Emphatic teaching and explanation to young persons, prohibitive measures against the production and consumption of alcohol, restriction of licenses, reform of public houses, a stand made against the prevailing thoughtless customs of drinking, the example of the educated classes, promise good results in this field. Certainly, too, much can not be done by an improvement in the general social and living conditions.

There belong to this group a series of soporifics, which may occasionally lead to severe nervous conditions. But since it is here a matter of very rare occurrences—after the continued use of paraldehyde, *paraldehyde* deliria, *veronal*—comatose intoxication-like conditions, with cardiac weakness, etc.—it suffices to call attention to these and, for the rest, to refer to the toxicological literature. The best known are the nervous toxic effects of the first artificially prepared soporific, *chloral hydrate*. The acute poisonings lead, after irritation phenomena of the mucous membranes, combined with paralysis of the cardiac and respiratory centers, to sopor and eventually to death. Chronic misuse causes general somatic phenomena, skin affections, erythema, purpura (even in acute poisoning), digestive disturbances, marasmus, pains in the limbs, ataxia, psychic depression, deliria. The treatment consists in enforced abstinence in an institution. These intoxications have become very rare.

(b) Sulphocarbonism (Poisoning from Bisulphide of Carbon)

Almost invariably, when being used in the rubber goods industry (vulcanization of the rubber) the inhalation of fumes, rarely moistening with liquid bisulphide of carbon, leads to intoxication. In addition to somatic disturbances through irritation of the mucous membranes, gastritis, catarrhs of the respiratory apparatus, there frequently result *neuritides* (see special chapter), which attack by preference the lower extremities, and cause a pseudo-tabes, not, however, associated frequently with bladder and pupillary disturbances.

Frequently the carbon bisulphide poisoning leads to *neuroses*, now of a neurasthenic-hypochondric character, pain in the nape and the temples, backache, pain in the limbs, tired feeling, sleeplessness, etc., now to hysteric phenomena, pressure points, anæsthesias, paralyses, contractures, etc. These functional disturbances are often found combined with slight organic symptoms such as changes in electrical excitability and slight muscular atrophies. According to the results of animal experimentation, these correspond probably to peripheral neuritic changes as well as to slight central changes in the nervous system.

Above all things, *psychoses* are often found as sequelæ of the poisoning. In light cases, there are brief conditions of simple psychic inhibition, light stupor, lasting for several days, at other times, we find transitory exaltations, like the *intoxication* following the acute poisonings; frequently there are found severe and long-continuing mental disturbances, in the origin of which, bisulphide of carbon plays an important ætiological rôle. The forms in themselves, are not characteristic, curable amentia, and mania, catatonia often ending in dementia; on the other hand, the development is frequently peculiarly influenced by the poisoning. The psychic diseases always require for their development a pronounced predisposition, and as a rule appear in a patient after a comparatively short period at work, the nervous diseases mostly only after months.

The **treatment** above all things, must insist upon immediate cessation of the injurious work, see to general strengthening, etc. The possibility of such diseases should be averted by prophylactic hygienic measures.

2. VEGETABLE POISONS

(a) Morphinism

A knowledge of chronic morphinism is of great importance to the physician, since the disease, as a rule, comes into existence as a result of physician's treatment. The restful sedative influence upon the nervous system exerted by morphine in medicinal doses, especially in so prompt a manner when subcutaneously injected, makes it a remedy fraught with blessings in physical or psychic pains or in sleeplessness, but it has this unpleasant trait—that after prolonged use of it, a habit is formed, which demands the use of increasingly larger doses to produce the same effect, and, when the use of it is discontinued, it evokes unpleasant, even critical disturbances, phenomena of abstinence, which can be got rid of only by a new dose of morphine. Now the phenomena of habit and of abstinence can be controlled and held within bounds as long as the physician undertakes to give the patient each of his injections, but from the moment when the patient, first to get rid of pains, later too, without this reason, gives the injection himself, only to get rid of the direful phenomena of abstinence or to evoke the euphoria conditioned upon the drug, is established the circulus vitiosus, which leads to injections of increasing quantities, and thereby to the phenomena of chronic morphinism. We speak of a chronic morphinism, wherever the chronic misuse of the narcotic has led to the necessity of continually taking it into the body. The number of morphinists is even now very considerable. It consists, naturally enough, for the most part, of physicians, druggists, their wives, nurses, and other persons in close association with the medical profession. Probably every person is susceptible to this poison, but psychopaths, degenerates, and congenital mental weaklings form a considerable percentage of morphinists, since they are less able to resist temptation, succumb more frequently to it, and often make use of morphine with the basest motives.

Morphine affects the nervous centers directly. As animal experimentation shows, it leads first to an increase, then to a decrease of reflex excitability. It causes pupillary changes (miosis). Larger doses paralyze the respiratory centers, cause lowering of the blood pressure, retardation of the pulse, probably through paralysis of the accelerators. Furthermore, the poison restricts the secretion of the mucous membrane and glands, inhibits the peristaltic motions and disturbs digestion. The latter is probably due to the elimination of the poison in the stomach. Part of the morphine, unchanged, leaves the body in the urine, part is transformed to oxidomorphine, a substance to the formation and secretion of which the phenomena of abstinence are traced back.

By occasional experiments upon human beings it has been shown that in small doses at first, there is an excitement of the imagination, of the intrapsychical activity, with deadening for outer impressions, decrease in pain sensation, and difficulty in the transformation of concepts into motor actions.

Symptomatology.—We can not enter here into the phenomena of the *acute* poisoning, which with increasing coma, narrowness of pupils, pallor and cyanosis of the skin, lowering of the pulse, of the respiration and bodily temperature, sometimes with epileptiform twitchings and spasmodic attacks, leads to death or after a long sleep ends in recovery.

In chronic morphinism we have to distinguish between intoxication and abstinence phenomena.

The *intoxication phenomena* appear after varyingly long individual use of the drug, after weeks or months or years, in unpleasant manner, preceded in the beginning, especially because of the dulling of the pains in the original trouble, by a condition of general euphoria.

In the foreground stand *digestive* disturbances, corresponding to the effect on the digestive tract (secretion of the morphine within it, inhibition of the peristaltic motions, of the secretion of muriatic acid and digestive juices). There is obstinate *constipation*, loss of appetite, a feeling of fullness in the stomach, nausea, vomiting, a special aversion to meat. The mucous membrane of the mouth is dry, an excessive flow of saliva occurs sometimes.

The skin, first of fairly good color, becomes pale, lustreless, rough, dry;

the secretion of sweat and sebum cease. Frequently there is an inclination to the formation of boils. Hyperhidrosis occurs also. Occasionally there are found trophic disturbances in the hair, nails, even in the teeth.

After long misuse there are found regularly *disturbances of the sexual functions*, loss of libido, in men impotence, absence of erections; in women, dys- and amenorrhœa, dryness of the vaginal mucous membrane. Conception occurs rarely, and then often leads to abortion, or pregnancy and childbed evince other disturbances.

Vasomotor disturbances are often found. The appearance of albumen in the urine, and intermittent fever, as a result of morphinism alone, are disputed.

There are early found extreme *pupillary narrowing* (miosis), with sluggish, insufficient reaction to light; accommodation paresis, and insufficiency of the interni sometimes occur. General muscular weakness, tremor, disturbances in coordination, and ataxia are rare.

The initial *psychic symptoms* are a slightly exalted euphoric mood, with stimulation of intellectual power, but inhibition of activity. Later too, intellectual capacity is usually retained permanently at a considerable height, but the power of decision and the emotional life suffer. With the habit of riding over the unpleasant things in life by means of a new injection, with the absence of normal fluctuations in feeling, with the slavish dependence upon the poison, the important interests in life are lost. An *indolence* appears, an indifference to the weal and woe of his nearest relatives, etc. If there are not always pronounced *ethical defects*, truthfulness usually suffers; especially relative to all questions concerning morphinism one must never trust the patient. In many patients, especially under the influence of slight abstinence phenomena, there occur even offenses against the penal law, theft, prostitution among women, deceit, and falsification of documents (falsification of prescriptions).

Psychic disturbances of circumscribed nature, attacks of terror, hallucinatory conditions, injurious ideas, are very rare, but usually with continued intensive use, there appears gradually a progressive physical decay, a *marasmus*, which may indirectly through the loss of resistance to intercurrent diseases, lead to death. Many morphinists commit suicide.

Often the patients attempt to wean themselves from the use of the poison, sooner or later, becuse of the many unpleasant phenomena. But the attempt succeeds only in exceptional cases, because of the *abstinence phenomena* that follow every abstention.

It is quite natural, too, that they should appear frequently even during the intoxication-period, often being suggested before almost every new injection, especially when the patients for certain reasons, such as difficulty in procuring it, etc., are forced for once to omit the dose, or to diminish it. There are more serious phenomena as soon as the morphine is accidentally or purposely removed entirely. The intensity here, too, changes with the length and strength of former injections.

Only a few hours after the last dose there appears a certain restlessness in the patients. The *reflex excitability* is considerably *increased*. There is frequent, almost spasmodically recurring, yawning, sneezing, coughing, retching and vomiting. Instead of the former inhibition of secretion, there is a more or less profuse secretion of the mucous membrane and the glands, in the nose, bronchial tubes, and alimentary canal. The latter, with increased peristalsis, leads to quite profuse watery, finally often bloody diarrhœas. Strangury is frequent and also bladder and biliary colics. The sexual function is again active; the appearance of frequent pollutions and erections, the return of the menses in women, usually accompanied by considerable discomfort, are usual. In the skin are shown numerous vasomotor phenomena, pallor, cyanosis, cold feet, rushes of heat to the head, etc.

The patients get worse rapidly. *The pulse rate rises*, the pulse is soft, dicrotic; in severe cases it finally becomes irregular, imperceptible and frequently there is a collapse.

Among other nervous phenomena, pupillary disturbances appear such as abnormal width, inequality, and accommodation pareses.

Pains of all sorts, in the back and in the calves of the legs, neuralgic pains in the region of the trigeminal, paræsthesias as well as ataxia, tottering gait and tremor, are regular phenomena.

In the *psychic realm*, there appears first a high degree of *restlessness*, *anxiety*, feeling of oppression; the patient tosses in bed, passionately demands morphine to obtain which he would use any possible method. Usually there is total *sleeplessness*, or restless broken sleep is had, only for a short time. Some patients lie totally apathetic, exhausted, and no longer react to anything. In many, hysteria-like *conditions of excitement* appear, more rarely deliria with visual hallucinations, dream-like failure to recognize their surroundings. Finally, delirium tremens-like states have been observed, with tremor, speech disturbance, possibly diplopia, also with albuminuria, besides anxious conditions of excitement and a strong inclination to suicide.

The duration and intensity of the abstinence phenomena depend upon the dose formerly injected and the manner of the withdrawal. Even when the disease runs a rapid course, some of its phenomena last for weeks after withdrawal. Dangers threaten in abstinence or withdrawal first through the appearance of cardiac weakness, collapse, and secondly through the possibility of suicide. For the rest the *prognosis* of morphinism depends on whether one succeeds in stopping the use of the poison and preventing recurrence from the renewed misuse of the drug.

The **diagnosis** is not always easy. Where the anamnesis is of no avail, the marks of injections, the abscesses and scars due to them are indicators. Occasionally the change in the nature of the patient after and between the injections is a suspicious symptom. There are found the characteristic phenomena (pupils, etc.) of morphinism. Eventually, the chemico-pharmacological proof of the presence of morphine in the urine must be tried.

Pathological Anatomy.—No marked and characteristic changes have been made known so far. Investigators have, however, experimentally, but not in man, proved the presence of changes in the cells. The recently described degeneration phenomena of the nerve fibres in the peripheral nerves and spinal cord are not yet of assured importance.

Treatment.—The only rational treatment of chronic morphinism consists in a systematically executed *withdrawal treatment.* Since the patients are only exceptionally able to carry this out themselves, it must take place under strictest supervision, preferably in an institution. Since, as we have seen, it is not without dangers, one will have to dispense with total withdrawal, when severe physical diseases, cardiac affections, etc., speak against it. This must also be done in many cases in which the original disease, especially in severe, painful and hopeless affections, makes it impossible to give up morphine.

According to the method of execution, one distinguishes between withdrawal and substitution methods. The latter, on the whole, however, are now abandoned, since the substitution remedies, cocain, codein, heroin, opium, etc., are more injurious than morphine, often leading to a new habit connected with the substitution drug, which is then frequently injected besides the morphine.

In the withdrawal methods one distinguishes a gradual, a sudden, and a rapid method. First physicians always tried to withdraw morphine gradually, and there are even now those who defend this process in a somewhat modified form. But much is to be said against it. It by no means spares the patient the abstinence phenomena, but protracts them considerably and thereby weakens the power of resistance in the patient. But above all, supervision is made very difficult, owing to the duration of the process, and a guarantee that the patient will not procure morphine could scarcely be given.

In the *sudden* method the morphine is stopped at one stroke. Then there often appear deliria, and very frequently collapses, which even when the physician is present, may threaten life, but nevertheless the method is comparatively certain, and the patients very rapidly recover from the withdrawal.

The method of so-called *rapid withdrawal* developed and recommended especially by Erlenmeyer, is now most frequently used. One immediately omits a very large part, a half to two-thirds of the dose last injected, which is usually excessive, and withdraws the rest rather rapidly in the course of 8 to 10 days. Even in this form, considerable abstinence phenomena appear, but the danger quoad vitam is only slight. The abstinence phenomena can be palliated in some particulars. Occasionally, it is of some assistance, if one in the place of morphine first gives dionin in like quantities, which may then be withdrawn in precisely the same way but apparently with greater ease. We have in morphine itself a means that in abstinence immediately lightens or removes all phenomena. This is of the greatest importance especially for the *collapse*, and here with one or more injections of 0.03 one can directly save life, even when such stimulants as camphor, coffee, etc., in large quantities, fail. Favorable also are the giving of alkalies, letting the patient drink great quantities of Fachinger water, possibly, too, washing out the stomach with it, or with plain water. Probably these measures produce their favorable results, by neutralizing the muriatic acid that is produced in excess. Vomiting may occasionally be checked by the use of ice and very cold beverages (milk). Alcohol often proves of avail against restlessness, but one should not give it too freely, since morphinism is easily replaced by alcoholism. The bromides are serviceable here also. Sleeplessness can be combated by soporifics, trional 1.0 to 1.5; veronal 0.5 to 1.0; it would be better to avoid chloral hydrate and paraldehyde, since they occasionally produce conditions of delirium. Lukewarm baths with cool showers and protracted warm baths, often prove helpful. For many of the troubles, even for sleeplessness, good results can be obtained through psychic influence, especially by hypnosis.

Unfortunately, even after the withdrawal has been successful, there is considerable danger of *recurrence*. A sufficiently long treatment, staying for months in an institution, is therefore always desirable. Of greatest importance, in morphine also, is prophylaxis. Morphine must be used with greatest care in chronic diseases as well as in simple nervous conditions. The injection of morphine should never be left to the patient himself. Former morphinists demand especial care, since in them, a single injection will often lead to a relapse.

(b) Cocainism

Cocain even in small doses exerts a sedative influence upon the nervous system, particularly upon its sensory elements, and an influence at first stimulating, later paralyzing, on the motor apparatus. It causes increase in respiration, contraction of the blood vessels, heightens the blood pressure and in large doses, has a paralyzing effect. In man after small doses, there are exhibited besides local anæsthetic effects, a peculiar euphoria, an increase in the activity of the mind. Large toxic doses lead to intoxication-like conditions, with depression, inhibition of thought, difficulty in speech, also to hallucinatory deliria and collapse, conditions, which after a long sleep, lead to recovery, or eventually amidst convulsions to death. The effect varies with the individual, also with the concentration of the solution. Occasionally in predisposed or physically weakened individuals protracted conditions of nervous weakness and terror, characteristic of an acute intoxication, follow one excessive dose (above 0.05–0.08).

Chronic cocainism rarely results from continuous external use of cocain as a local anæsthetic for the mucous membrane of the nose, pharynx, etc. In most cases it develops on the basis of morphinism, from an attempt to withdraw morphine by the substitution of cocain, which, in a certain respect, works in opposition to it. The result is always that cocain continues to be injected in addition to the morphine, and that too in considerable doses.

Cocain exhibits a number of very unpleasant peculiarities. Its effect quickly passes away, and the patient is therefore forced to take larger and larger doses. Cases in which 5–8 gr. were injected are not rare. Besides this it produces, and in far more pronounced degree than morphine, *a passion for the drug*, not conditioned upon abstinence phenomena, but as a very irresistible impulse towards the poison.

Symptoms.—Even after a short time, there is loss of appetite, disturbances in nutrition, emaciation, pallor, trophic changes in the skin and nails, edemata. There appear also flaccidity of the vessels, acceleration, rarely retardation of the pulse, dyspnœa. The sexual functions are impaired. There are found azoospermia, impotence, amenorrhœa.

The pupils are usually dilated. The musculature becomes flaccid and weak, the gait tottering and infirm. The patients display trembling in the hands, in the musculature of the face, hesitating uncertain speech, frequently cramps in the calves; in some cases there are pronounced epileptic attacks.

In the *psychic realm* a decrease in intellectual capacity is very soon noticed, absent-mindedness, forgetfulness, a very characteristic circumlocution in speaking and writing. To these are added restlessness, excitement, conditions of fear. Sleep is always insufficient. The *lowering of the moral qualities* and of the higher feelings is striking. The patients lose interest in their vocation and family and become untruthful and careless in appearance and behavior. They are irritable, discontented, querulous, suspicious.

Pronounced psychoses of the nature of acute hallucinatory insanity are common. There are hallucinations affecting all the senses. The patients hear people speaking about them, shooting, see persecutors, smell poisonous gases, taste poisons. Especially characteristic are certain often *combined hallucinations of sight and touch.* White surfaces appear to the patient perforated, they observe in their skin countless often microscopically small objects, worms, crystals, cactus thorns, etc., which they attempt to scratch and cut out of their skin.

They believe that they are persecuted, watched, taken to task for their cocainism. The hallucinations are frequently of sexual content. They also see obscene pictures, hear themselves enticed to coitus. Frequently a genuine *jealous mania* is found as in drinkers, and the cocainist does not shrink from actions that are dangerous to others.

The outcome, unless a halt is made in time, is unfavorable. Marasmus appears. Suicide is not infrequent.

Treatment.—The only method indicated in all cases—withdrawal of cocain—is in itself very easy in contrast to that of morphine, especially because it brings with it virtually no unpleasant phenomena of abstinence. Cocain may be stopped immediately without any further thought. Usually only then follows the difficult withdrawal from simultaneous morphinism (cf. above).

The hallucinations usually disappear very quickly. On the other hand, the correction of the obsessions, and insight into the disease often follows only after months. For this reason, as well as because of the frequent general danger, a sufficiently long sojourn in an institution under strict surveillance is necessary. Only this can guarantee against the frequent relapses that otherwise occur.

(c) Nicotinism

Acute poisoning by nicotin, with vertigo, syncope, retardation of the pulse, diarrhœa, vomiting, well known from the "first cigar," may in severe cases (rectal injections of tobacco), cause sudden death through paralysis of respiration, but we are concerned here only with chronic poisoning, which usually results from excessive smoking of strong cigars, cigarettes, and tobacco.

Symptomatology.—Besides catarrhal phenomena in the pharynx and bronchial tubes, loss of appetite, feeling of fullness, vomiting, diarrhœa, there are found general nervous phenomena, pressure in the head, sleeplessness, vertigo, tremor, but especially cardiac symptoms, palpitation of the heart, "stitches" in the region of the heart, irregularity and jumping of the pulse, even when organic changes in the heart and blood vessels, arteriosclerosis, myocarditis are not present. Phenomena on the part of the eyes, amblyopias with avoidance of light, subjective light phenomena, central scotoma, are common; cerebral hemorrhages, neuritis optica, i. e., papillitis are rare. Finally, there are found psychic disturbances, general exhaustion, depressive states, a high degree of irritability, subjective noises, obsessions, rarely psychoses of the nature of a subacute hallucinatory persecution mania.

Treatment.—The most important indication is the immediate withdrawal of the nicotin. Pronounced abstinence phenomena do not usually appear upon cessation of smoking. Rest, sojourn in the fresh air, hydrotherapeutic procedures, sitz-baths with showers, carbonic acid baths, lying in the open air, etc., accelerate recovery, especially when pronounced arteriosclerosis (intermittent claudication, myocarditis, etc.) has not yet appeared.

Smoking must be given up altogether, or in light cases, be very much restricted. Smoking upon an empty stomach, inhalation of smoke, are above all forbidden. The so-called "nicotinless" cigars, are to be permitted only in restricted quantities.

(d) Ergotism

The *rather rare poisoning* by ergotized grain arises, and mostly in epidemic form, from taking into the body the grain or flour infected by ergot (mycelium of claviceps purpurea). Since, in the course of time, its active substances decrease (cornutin, sphacelin acid, sphacelotoxin), the effects of it are most intensive in July and August, and the epidemics usually break out in this season. Cases as a result of medicinal administration are rare, not so much because of the preparations, as because of the unequal therapeutic value of the ergotin itself.

Symptoms.—*Acute* poisoning is accompanied by violent gastro-intestinal phenomena, a cool, cyanotic skin, small pulse, formication over the entire body, muscular weakness, possibly too, headache, vertigo, dilatation of the pupils or miosis. There result deliria or sopor and the outcome is fatal or a very slow convalescence.

In *chronic* poisoning two forms are usually distinguished:

1. *Ergotismus gangrænosus*, with ischemic anæmia, and finally dry gangrene of the more or less distal parts of the extremities or nose, ears, etc.

2. *Ergotismus convulsivus*, the real nervous form. The disease picture is here too introduced by gastro-intestinal symptoms, diarrhœas, constipation, excessive thirst, voracious hunger. The most various sensations appear, tinnitus aurium, headache, vertigo, scintillations before the eyes, pains in the back and limbs, and the virtually pathognomonic symptom, an exceedingly unpleasant tickling in the fingers, toes, trunk, chest and back (*formication disease:* Kriebelkrankheit). To these are added intermittent *convulsive-like phenomena*, tonic-clonic muscular twitchings, especially flexing spasms (claw-hand), cramps in the calves, tetany and chorea-like attacks, empros- and opisthotonus, finally often epileptic attacks, usually those that are major and developed, more rarely, petit mal. Sensory disturbances, *paræsthesias* in the extremities, anæsthesias, diminution in the feeling of pain with the girdle sensation, lancinating *pains*, ataxia, finally above all, the *loss of the tendon reflexes* complete the picture which has been designated as *ergotin tabes.*

In a very large number of cases, there are combined with it *psychic disturbances*, stupor, extreme sleepiness, coma, conditions of confusion, melancholic depression, and feeling of incapacity, finally dementia. Maniacal outbreaks are rare. The frequently long retention of the feeling of illness is characteristic.

Chronic ergotinism does not, like the acute form, lead to abortion, but it exerts a detrimental influence upon the progeny. In them, degeneration, idiocy, epilepsy, are exceedingly common.

The **diagnosis** is evident from the peculiar grouping of the symptoms, from the course, if one thinks of the ætiology. Bread that contains ergotin looks dark, sometimes even bluish black. The presence of ergotin in the contents of the stomach and food can be demonstrated.

Pathological Anatomy.—Gray degeneration of the posterior columns of the spinal cord, as in tabes, besides changes in the cells and vessels, and hemorrhages into the gray substance are present.

Treatment.—Immediate discontinuance of the injurious food, strengthening nourishment of the patients who are mostly emaciated and in a wretched state is imperative. To be recommended at first are laxative remedies, calomel, later astringent, tannin, opium also as sedative, chloral hydrate for the spasms and convulsions. The formication is best treated by warm baths. Prophylaxis demands control of the State over the impure flour and grain, and strict prohibitions against its use. Even in the medicinal administration of ergotin itself, one must be careful because of individual susceptibility, especially since the effects of the preparations are so variable.

(e) Pellagra

Pellagra occurs almost exclusively in those countries (Italy, Spain, Austria, Tyrol, Friaul, Bukowina, etc., also in Africa and America) in which Indian corn (maize) is almost the only article of nourishment of the country population, and is found only in the country, not in cities. The disease probably rests upon an intoxication, evoked by eating moist corn spoiled by fungi (aspergillus or penicillium species). The toxins produced by the above mentioned hyphomycetes seem to play the essential rôle, but probably also the injury to the constitution, especially the tendency to intestinal diseases, as a result of one-sided, insufficient nutrition in the polenta eaters is of importance.

Symptomatology.—From the predominance of nervous and psychic symptoms, pellagra has also been designated psychoneurosis maïdica. Of the usual *somatic* phenomena, often decisive for diagnosis, we must mention:

1. *Skin phenomena*, erythemata, especially upon the uncovered places of the body, with pink somewhat raised edge, which heal with scales, thickening of the skin, later atrophy and deposition of pigment. Diffuse hyperpigmentation also is not rare.

2. *Gastro-intestinal symptoms*, diarrhœas without fever, mostly without pains, occasionally of dysenteric nature, dryness of the mucous membranes, furred tongue with exfoliation of the epithelium, loss of appetite, flow of saliva, etc.

3. General *cachexia*, wasting of the glandular organs, muscles, brittleness of the bones, etc.

Of *nervous symptoms*, there appear first those of functional nature, to which later organic phenomena are gradually added. Thus are found *subjective troubles*, pains in the nape, head and back, paræsthesias in the limbs, fatigue, vertigo, visual disturbances, roaring in the ears, sleeplessness, loss of appetite, violent hunger, disturbances in swallowing, transitory pareses in the muscles of the face and eyes, pallor, cyanosis.

Muscular irritation phenomena frequently appear during the course of the disease. Cramps in single muscles (calves of the legs), tonic tension but also myoclonus-like phenomena, fibrillary and fascicular-like twitchings, increase in the mechanical and electrical excitability, Trousseau's phenomenon. There are *pareses* especially in the extensor musculature, spasms in the flexor musculature, the gait becomes spastic paretic, finally there are found contractures, in the lower rather than in the upper extremities. Besides general muscular weakness, there are genuine degenerative paralyses and total atrophy. Co-ordination disturbances, Romberg's phenomenon, ataxia of the arms rather than of the legs, are not rare. Otherwise, paræsthesias predominate in the sensory realm, usually in the touch, thermic and above all in farado-cutaneous sensibility.

The *tendon reflexes are usually increased*, rarely lost, the cutaneous reflexes mostly weak, but Babinski's phenomenon appears early. Usually the lower extremities are more severely attacked; there are frequently found differences between the right and the left side. Pupillary disturbances appear mostly only in the terminal stages. There is paralysis of the sphincter only in very severe cases.

Of cerebral disturbances, the *pellagrous attacks* are important, corresponding to the picture of Jacksonian epilepsy, more rarely to genuine epileptic attacks, chorea or tetany.

Psychic disturbances are found almost regularly, and even in the early stages, first mostly as a slight retardation of all functions, slowness of thought and judgment, lack of concentration, motor inhibition, depressive, melancholy or hypochondriac moods, irritability, etc.

In more severe cases there are more or less pronounced psychoses. On the whole, depressive conditions predominate with a feeling of incapacity, apathy, bewilderment, but also conditions of confusion, hallucinatory deliria with energetic motor reaction, severe dimmings of consciousness, as well as more independent diseases and intercurrent episodes occur. Catatonic disturbances in movement and catatonic phenomena, immobility, mutism, etc., are not rare, nor are genuine catatonias ending in dementia. Pure melancholy and mania are rare, progressive paralysis of the insane on a pellagrous basis alone does not occur, though certain final stages may bear a close resemblance to that disease.

In cases running a chronic course, but also in cases in which the development from the beginning is acute, there may come to be extremely dangerous conditions of confusion, excitement and prostration, like the delirium acutum, but frequently without any sign of fever. Because of the often accompanying diarrhœas this disease is frequently called *pellagra typhoid*. It has nothing in common with typhus abdominalis.

Course.—Pellagra is usually a chronic disease with remissions and intermissions. It usually begins in spring, retrogresses in summer, then exacerbates in the autumn, only to reappear after a winter's remission in the spring, in increased degree, and with more severe phenomena. There is no absolutely regular sequence of phenomena, though in general there appear first cutaneous, gastro-intestinal and functional nervous symptoms, later organo-nervous, cerebro-spinal and psychic phenomena, finally cachexia and dementia. Important symptoms, even those on the part of the skin may be lacking (pellagra sine pellagra), etc. At any rate, the course without treatment at the proper time ends in death.

The **diagnosis** in the typical cases is easy; anamnesia, ætiology, erythema, the grouping of the nervous phenomena, functional, convulsive, spastic-paretic, sensory disturbances, psychoses, present a characteristic picture, but numerous abnormal and abortive forms occur.

The **prognosis** depends upon the development of the phenomena. Not only the initial functional symptoms, but also the spinal, spastic paresis, etc., may retrogress almost to complete restoration. The psychoses may in large measure be cured. Only when it has progressed to cachexia, severe paralyses, and atrophies of degenerative character can no considerable improvement or recovery be hoped for.

Pathological Anatomy.—Beside diffuse, non-characteristic organic changes, mostly of atrophic nature, there are found organic changes in the nervous system. The nerve cells early display changes, swelling, extensive degeneration, sclerosis. Besides, there is degeneration of the medullary sheaths and of the axis cylinders. In later stages, there are found in the spinal cord characteristic changes, gray degeneration especially of the posterior and postero-lateral columns, which, however, does not restrict itself to the boundaries of the systems, and is especially pronounced in the lumbar and cervical portions of the cord.

Treatment.—In the first place, change in diet, more varied especially as to vegetable food, improvement in the state of nutrition, rest, removal to suitable hygienic surroundings, placing in suitable institutions are all important. Medicinal treatment, mainly with arsenic, is of less avail. Attempts at treatment with an antitoxic serum may perhaps in the future bring about the desired result. Especially important is prophylaxis, change in diet, planting of other grains, introduction of pure grain, instruction of the people, raising of the general hygienic and social conditions. The task is all the more urgent inasmuch as pellagra in the parents seems to create in the children a certain hereditary predisposition to pellagrous diseases.

Similar diseases may occur occasionally in Southern Europe, from eating other plants, *lathyrismus* (chick pea, lathyrus sativus), but it must suffice merely to mention this.

3. POISONING BY THE METALLOIDS

(a) (Bromism)

The long continued absorption of bromine leads to intoxication usually only when used in the form of bromide of sodium, of potassium, of lithium, of ammonium; hardly ever when taken in organic combination with fats (bromipin), (bromalin), albuminous substances (bromeigon, etc.). The bromine is accumulated in considerable quantities in the organism, crowds out the chlorine from its compounds in the fluids of the tissues and the individual organs, and leads to disturbances mainly on the part of the skin, the general nutrition and the nervous system, which one should unquestionably know when using it therapeutically.

Symptomatology.—Usually very soon, but the time varies with the individual, there appear *exanthemata*, usually in the characteristic form of bromide acne, pustules without an area of inflammation, the purulent content of which contains bromine, occasionally small abscesses with spongy, indolent granulations. *Catarrhs* of the mucous membranes of the respiration and alimentary tracts loss of appetite, coughing, expectoration, lowering of the nutritive condition, and *cachexia* are frequent.

Bromine causes a *considerable lowering of the excitability of the central nervous system*, fatigue, muscular weakness, lowering of the sensibility, but also occasionally pains in the limbs, pressure in the head, forehead, and temples, *weakening of the reflexes* in the skin and mucous membranes (conjunctival-pharyngeal reflex); the patellar tendon reflexes also may disappear altogether. There appear furthermore disturbances in swallowing, mumbling speech, tottering gait, loss of the libido sexualis. The *psychic processes*, also, are *impaired*, understanding, reproduction, association, judgment. Real dementia arises as a rule only from the diseases treated with bromides, especially epilepsy. With continued use, there may appear disturbances in the activity of the heart, small, frequent, irregular pulse, morbid desire for sleep and coma, and, through disturbance of expectoration even aspiration pneumonia and death.

Diagnosis.—With the anamnesia given, the diagnosis is easy; when using the bromides therapeutically, one must think of the danger of intoxication. For the rest, the characteristic bromide acne and the proof of the presence of bromine in the urine are of assistance.

Treatment.—Withdrawal of bromine, which eventually by the consumption of salt in large quantities, may quickly be removed from the body (care must be taken in epilepsy). Otherwise, use of stimulants, coffee, camphor, baths with cool showers, etc.

(b) Carbonic Oxide Poisoning

Origin usually through inhalation of mixtures of gases containing Co, illuminating gas, coal fumes, etc. The acute poisoning acts essentially through changes in the blood, but also exhibits predominantly nervous symptoms, vertigo, headache, rush of blood to the head, flickering before the eyes, buzzing in the ears, feelings of anxiety or pleasurable sensations, loss of consciousness, spasms and paralyses. Survival of the acute poisoning is often followed by consequent diseases, of which beside long-lasting psychoses, cerebral *hemorrhages*, especially in the classic place in the central ganglia, with their corresponding phenomena are of special interest. Also a chronic carbon monoxide poisoning with general nutritive disturbances, anæmia, fatigue, depression, possibly with spasmodic attacks occurs.

The treatment besides getting rid of the injurious substance and the plentiful supply of oxygen, has to follow the general rules that hold good for the morbid conditions in question.

There should be added here poisoning with certain substances, that are virtually nerve poisons, such as arsenic, but in respect to these, we refer to the corresponding chapters. The poisonings in the next group are closely related, but shall only be mentioned, inasmuch as they besides neuritis, cause other diseases of a nervous nature.

4. POISONING BY METALS

(a) Lead Poisoning (saturnism, plumbism).

All that has been necessary to mention in regard to the peripheral affections, the characteristic paralyses, the pains, which appear in this vocational disease and virtually are to be regarded only as the expression of neuritis, has already been stated in the discussion of the various forms of neuritis. Besides the neuritis, central changes in the nervous system obviously play a certain part, but they also occur independently. Lead has a direct toxic influence upon the nervous parenchyma, it can frequently be proved to be present in considerable quantities in the brain. Besides this, it produces, by means of circulatory disturbances, edema of the meninges and hydrops of the ventricles; it acts also by means of metabolic disturbances (of the nature of gout, and as a result of kidney diseases, of the nature of uræmia), and by means of changes in the vessels (apoplexies), thus acting indirectly upon the nervous system.

These rather different central affections are usually collected under the name *encephalopathia saturnina.*

Symptomatology.—Certain forms are specially remarkable, running a very acute course in a few days, the main phenomena of which are severe disturbance in consciousness, even to *coma*, usually fully developed *epileptic* *attacks*, appearing singly or in large numbers, and *deliria*, in which stupor and extremely confused excitement recur repeatedly and are momentarily interchanged. All these phenomena may appear isolated or in various combinations. Death is very frequent, in other cases recovery sets in after a few days, often however only after a condition of weakness that has lasted for weeks and months.

Frequently there develop on the basis of lead poisoning, *neuroses* of neurasthenic or even hysteric character, in the causation of which, a concomitant alcoholism often cooperates. There may exist besides individual neuritic symptoms, neuritis optica, disturbances in the eye muscles, etc. A weakening of the psychic faculties, simulated by dimming of the consciousness may recall the picture of dementia paralytica. Progressive paralysis of the insane is, it seems, occasionally modified in a peculiar fashion by simultaneous lead poisoning (atypical paralysis). In *epilepsy*, doubtless in many cases, lead poisoning has great ætiological importance, leading to typical grand attacks as well as to "absences," finally often to *psychoses*, above all to severe confusional states of epileptic character. There have also been described psychoses of the nature of a catatonia, of a chronic hallucinatory insanity.

Diagnosis.—Recognition depends upon the grouping of the symptoms which is occasionally in itself characteristic. The anamnesia and the proof of lead poisoning usually derived from the blue line on the gums, colic, typical paralysis, etc.

Treatment.—Immediate removal of the opportunity of absorbing the poison, transportation to places with suitable hygienic conditions, rest, warm baths (sulphur baths), care for evacuation of the feces. In the acute forms, lumbar puncture occasionally seems to be useful. After recovery from the intoxication, all further contact with the poison is to be avoided, the vocation must eventually be changed, since otherwise recurrence may be expected. In certain conditions it may appear even without new intoxications.

(b) Mercury Poisoning (Mercurialism)

Here, too, the chronic, mainly occupational intoxications, may lead to nervous disturbances, tremor, irritability, sleeplessness (erethismus mercurialis), occasionally, too, to confusional states with sense delusions.

XII

OPERATIVE TREATMENT OF NERVOUS DISEASES

BY

FEDOR KRAUSE (Berlin)

1. DISEASES OF THE BRAIN AND ITS MEMBRANES

Entrance into the brain is effected by opening the cranial cavity, namely by trephining. The introduction of antiseptic treatment of wounds has caused in this as in other fields, a thorough transformation. Our present day aseptic methods afford such great security that one may say that the mere operative act is almost devoid of danger. There is great danger, however, if the cranial opening is made too large, as in too extensive craniectomies. Then collapse and hemorrhage, especially from the diplöe, may threaten life.

The classical instrument for opening the cranium, the trephine, is now but little used, and then only in those cases in which a definite circumscribed opening in the skull-cap seems for the first sufficient, as for instance, in order to effect drainage of the lateral ventricle in hydrocephalus internus or for injecting tetanus antitoxin into the cranial cavity. But even for such purpose, we have now, in *Doyen's* burr, a more serviceable instrument. In large openings, in my opinion, the bone should be retained, if possible. *Wagner-Königshütte* deserves the credit of having first carried out the osteoplastic method in man. The nutrition of the flap, composed of both soft parts and bone, is effected through a pedicle that usually occupies the entire breadth of the flap, and is placed in a general direction towards the base of the cranium. The direction of incision may be seen from the corresponding illustrations in the text. Except in rare cases, I use this osteoplastic method in all openings of the cranium.

Since in large trephinings, the loss of blood from the soft parts may become very considerable and dangerous, I place a surrounding mass ligature in a rectangular area which includes the future flap, upon the outer side of the incision and also beneath the base of the flap, according to the precedent set by *Heidenhain.* After the operation is completed, the flaps are replaced, and the edges of the wound exactly approximated and well sutured. I remove all ligatures before placing the bandages upon the wound.

Once the cranial bone is stripped of its periosteum in the direction in which it is to be opened, some holes are bored to the dura mater. For this we use *Doyen's* instruments, which are operated by hand. No electromotor is needed either to cut around the bone; in my experience one always succeeds with *Dahlgren's* forceps, the model of which I have altered. Its great advantage lies in the fact that one is independent of the thickness of the skull, which often even in small areas, exhibits great differences, and therefore the dura is not exposed to the danger of injury, provided one separates it from the lamina vitrea before the bone is severed. For this I use *Braatz's* probes, which are made in three degrees of curve.

With the excision of the osteoplastic flap, without opening the dura mater, the first stage is over. Operations upon the brain are frequently radical and of immediate danger to life, so that it is always well to perform the operation in two stages, provided that no disadvantage follows from this procedure. In cerebral abscess, therefore, one will perform the entire operation at once; but even in most cerebral tumors, a delay of 8 to 10 days before extirpation does no harm; finally in epilepsy, where a disease that lasts for years is concerned, the operation may without doubt be performed in several stages. No rules of general application can be given. Occasionally impending collapse necessitates a cessation of the operation after trepanation; the further proceeding, in most cases, is optional with the surgeon—then it is a matter of judgment, whether or not to proceed. I have frequently seen in brain operations the condition of the heart and the pulse remain favorable for a long time, and then suddenly, even without loss of blood become worse; when once such a collapse has occurred, not even immediate halt in the operation can always avert death.

It is not the mere act of trepanation that exerts such a serious influence upon the organism. In other parts of the body, we perform far more extensive and bloody operations, without a similar collapse. With the wide opening of the cranial cavity, the intracranial pressure relations are completely changed, and such a change can not take place without exerting an effect upon the condition of the heart, blood pressure, and respiration. Thus the severe phenomena of shock which unfortunately occur all too frequently in operations on the brain are to be explained.

The question as to how much time should elapse between the two stages of the operation, can not be answered in general; it depends upon several circumstances. First, it depends upon how quickly the patient has overcome the consequences of trepanation; furthermore, upon the nature of the disease that one is trying to remove by the operation. An interval of 6 days is, according to my experience, the very briefest that should be chosen. Even then, one still sees occasionally, that after removal of the clinging coagulated blood particles, the old cut surface on the bone bleeds with considerable severity. It is better to wait from 9 to 14 days; even after so long a time, one can tear the fresh cutaneous scar with a blunt instrument. One finds the cavity of the wound filled with clots, which may cling so closely to the dura that they can not be removed by pledgets, but must be taken away with a sharp spoon.

For the *opening of the dura mater*, I have long since given up the cruciform incision, with the formation of four tips; this procedure considerably restricts the space in which the brain is to be exposed. I rather prefer a rectangular flap, the base of which I do not always place below, nor always corresponding to the base of the osteoplastic flap. If for instance one has to proceed forward to the vicinity of the sinus longitudinalis, it is much more to the purpose to form the bridge of the dural flap towards the middle line. Now, as a rule, the surface of the brain is exposed. The surgeon generally does not see the arachnoidea, the tissue of which was opened together with the dura; it appears as a separate layer only when it is edematously infiltrated or thickened by inflammation, *leptomeningitis chronica*, changes which we find especially in epilepsy.

The surface of the brain which appears in the operative area is always covered by the pia mater; both are for the surgeon inseparable structures. Removal of the pia would bring about immediate destruction of the upper layers of the cerebral cortex, and furthermore, through interruption of the circulation, produce far-reaching disturbances in nutrition, and therefore must, under all circumstances, be avoided.

Cranio-cerebral Topography

It is of greatest importance for the surgeon to know exactly the relations of the surface of the skull to the convolutions of the brain. We are afforded the quickest orientation upon the skull, when we mark the situation of the Rolandic and Sylvian fissures; the simplest method to accomplish this is by *Krönlein's construction*, Fig. 234.

Kocher thinks it better to determine the precentral fissure, and to deduce from it the orientation upon the surface of the skull. For his method see Fig. 235, which at the same time represents the sites for *Neisser's* punctures. None of the methods, however, can claim complete reliability in every case; and for this reason we are still forced to-day always to make the trephine openings quite large.

Cysts of the Brain and its Membranes

Traumatisms as well as inflammations play a rôle in their origin. Because of the well known elasticity of the cranial cap, the injury need not lead to a splintering or a breaking of the bone; on the contrary, this may be driven violently against the surface of the brain and then return to its own position, so that not the least change in it can be perceived in the X-ray picture or in the operation that becomes necessary. On the other hand, there arise in this manner contusions and hemorrhages of the brain and its membranes, which then in their further course occasionally cause the formation of an arachnoid cyst.

The products of decay formed as a result of encephalitis, can, unless they are absorbed, evoke the most severe phenomena, so that one may even be led to suspect a new growth. In especially fortunate and rare cases, the puncture alone and the withdrawal of the detritus, clear up not only the diagnostical doubt, but induce through absorption a final disappearance of the remaining waste matter and thereby insure immediate recovery. Cysts upon a meningo-encephalitic foundation, either cortically or subcortically located, have been frequently found by me in infantile

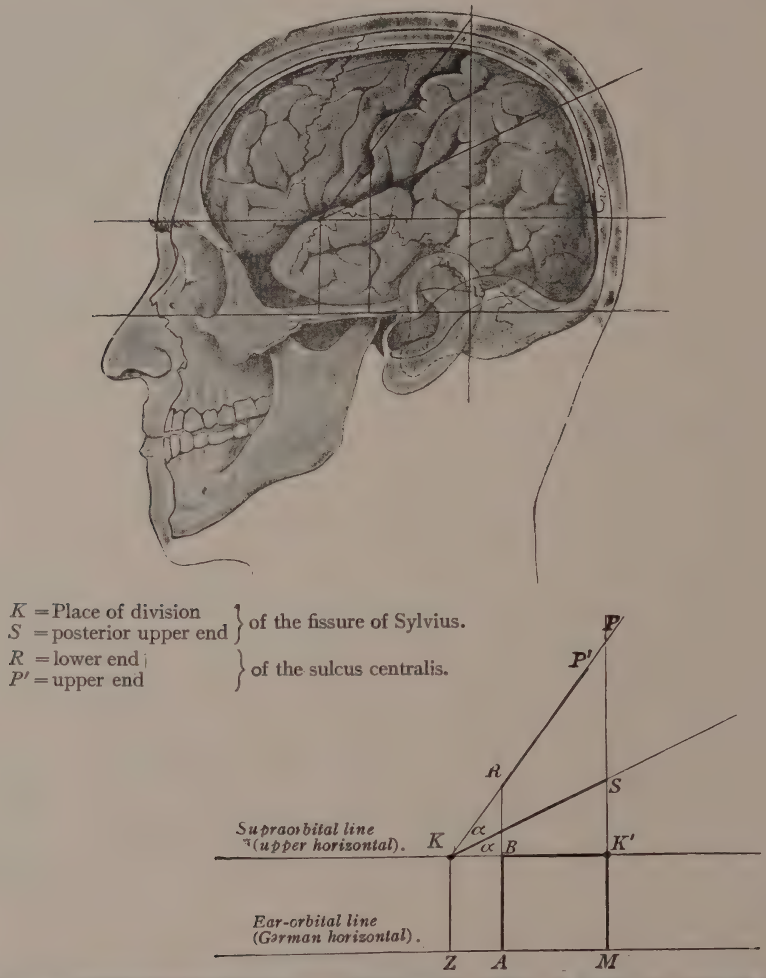
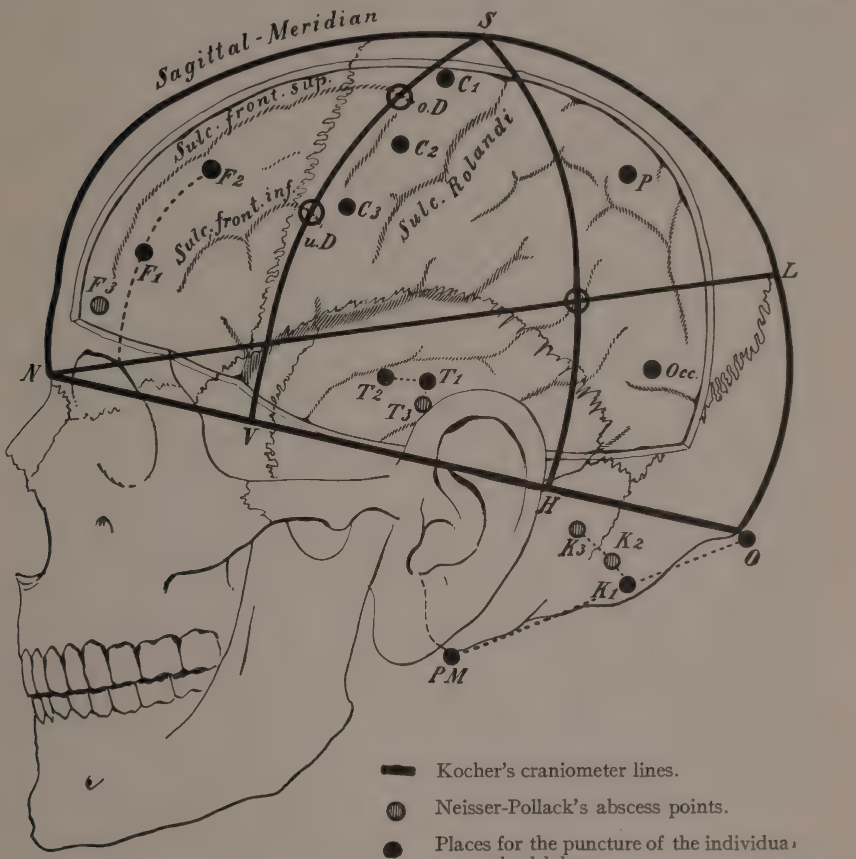


FIG. 234.—Kroenlein's construction, to determine the position of the sulcus centralis and of the fissure of Sylvius in the cranial surface. (*From Kronlein in v. Bergmann-Bruns Handbuch der praktishen Chirurgie.*)

cerebral paralysis, when, because of the synchronous *Jacksonian* epilepsy, I performed trepanation.

Furthermore some forms of tumor, especially sarcomata, tend to degenerate into cysts, and occasionally to form large isolated cysts, and in



▬ Kocher's craniometer lines.

▨ Neisser-Pollack's abscess points.

● Places for the puncture of the individual cerebral lobes.

S = Parietal point middle between *N* (root of the nose) and *o* (protuberantia occipitalis externa).
NVHO = Equatorial or basal line.
NSO = Sagittal meridian.
SV = Anterior oblique meridian, precentral line.
SH = posterior oblique meridian, linea limitans.
NL = Linea naso-lamboidea, between *SV* and *SH* designated by Kocher as linea temporalis *I*
o.D = upper, *u.D* = lower } third part point of the precentral fissure.
PM = apex of the mastoid process.

FIG. 235.—(*From Neisser and Pollack, Die Hirnpunktion. Mitteilungen aus den Grenzgebeiten,* 1904, *Page* 823.) In the scheme for the determination of the craniocerebral topography (according to Poirier-Kocher) the Neisser-Pollack puncture places are marked.

their vicinity, to create cavities filled with thin blood; and finally there are found in the brain parasites like the cysticercus and the echinococcus. In many kinds of cysts no cause at all can be established.

The treatment of the cysts varies according to the nature of their walls, which, in turn, depends upon the cause of their origin. In all forms in which the cyst-wall is composed of connective tissue and its transformation products, we attain a cure, if after complete removal of the contents, the cavity remains permanently empty. Puncture and withdrawal of the contents by no means always suffice; especially when the walls are infiltrated and thickened, as frequently happens when fluid has been collected for a long time even in the brain, this method will work only for a short time, since the rigidity of its surroundings prevents the collapse of the cavity. The safest procedure is opening by means of an incision, with consequent drainage or tamponade, in order to cause the walls to shrink and the cavity to become obliterated.

In the parasitic cysts—cysticercus and echinococcus—the extirpation of the sac is the safest procedure and should be performed if possible. Cysticercic sacs scarcely attain a large size, and therefore in general, one will be able to enucleate the sacs in those cases at least in which operatively approachable parts of the brain are affected. But in the echinococcus, even in the convex area of the cerebrum, the enucleation of the cyst en masse may fail because of its size and great extent; then tamponade is the ultimate resource. One would thus have to place the incision into the cyst in such a way that it would be separated from the rest of the wound, and therefore if it were necessary, could be treated separately by irrigation or injection of dilute *Lugol* solutions of iodine—iodide of potassium.

In the cerebellum, cystic degeneration of solitary new growths are no rarity, and here the diagnosis of simple or serous cysts should be made only if exact microscopic examination of the wall has given no hint of tumor tissue. For it has been established a number of times, that smaller or larger tumor nodules were present in the wall of the cyst. In all cases in which the cyst presents only a secondary degeneration of tumor tissue, the thorough removal of the entire new formation far down into the sound substance of the brain is absolutely essential. Furthermore, there occur cysts in the cerebellum which owe their origin to a sac-like protrusion of the fourth ventricle; then a connecting path is present between the ventricle and the cavity. The protrusion which is at first slight is gradually increased, and finally the cystic widening may extend far out from the vermis into the hemisphere.

Finally there have been observed in the cerebellum dermoid cysts, though very rarely.

Tumors of the Cerebral Membranes

which emanate from the inner surface of the dura mater, the arachnoidea or the pia mater and proliferate into the brain, are histologically speaking comparatively benign; it is usually a question of fibromata or fibro-sarcomata. In any case, they grow into the central organ, and produce all the phenomena of intracranial increase in pressure. But on more exact observation, they are seen only to push the substance of the brain before them, and form in it a cavity, the base of which is covered by an extremely thin layer of the corticalis, while the tumor itself is usually covered by a more or less conspicuous capsule. There may even be a small cavity between the two.

The external surface of the dura is rarely affected by the growth of the tumor, which in these cases virtually without exception takes place in the direction of the brain; it is then stimulated to chronic inflammatory proliferations, and since the dura mater possesses at the same time the functions of the inner periosteum of the cranial cap, the bone is also incited to new formations.

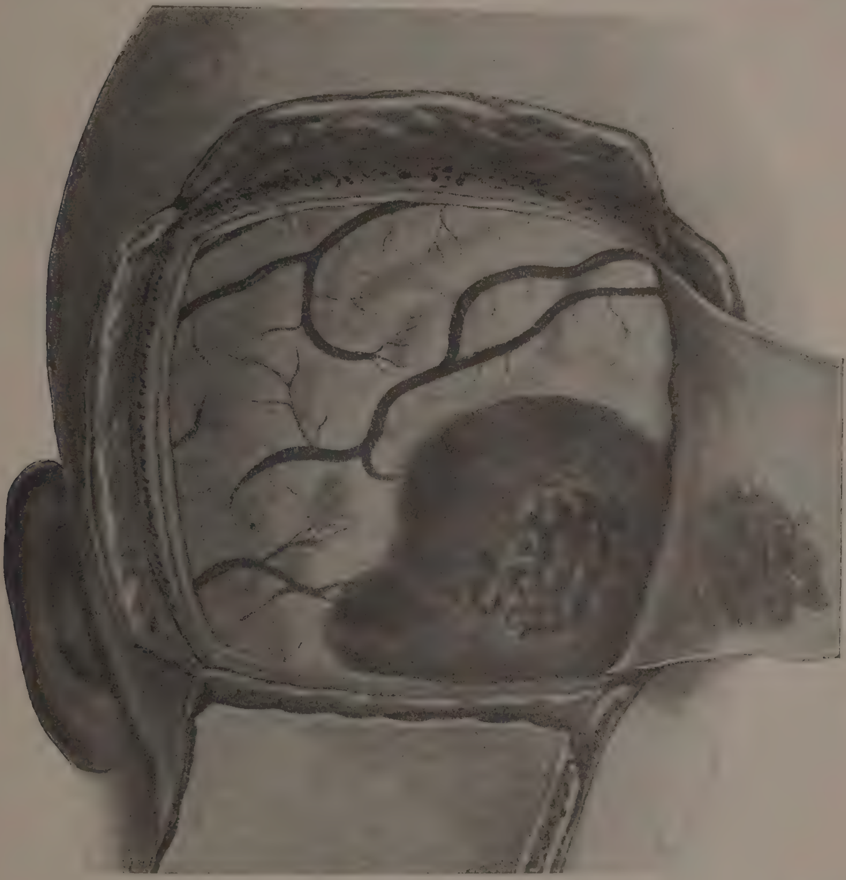


FIG. 236.—Fibrosarcoma of the occipital brain, arising from the inner dural surface. The patient, 35 years old, is entirely cured; since 3 years even the right sided hemianopsia has disappeared.

Only rarely are tumors found so detached in the brain that they spontaneously follow the pull upon the dura mater. They are nearly always tightly adherent to its inner surface and the incised dural flap can not easily be turned back. Then, in order to make possible a full view of the surface of the brain, and the extent of the tumor upon it, it must be dissected out at the proper place with knife or scissors, or, if feasible, be loosened with a blunt instrument, whereupon naturally, residues of the tumor are left behind upon its inner surface. The process is intelligible without further explanation, from Fig. 236. One recognizes immediately from this example how much more advantageous incision into the dura forming a flap is, than the formerly employed cruci-form incision resulting in the formation of four tips.

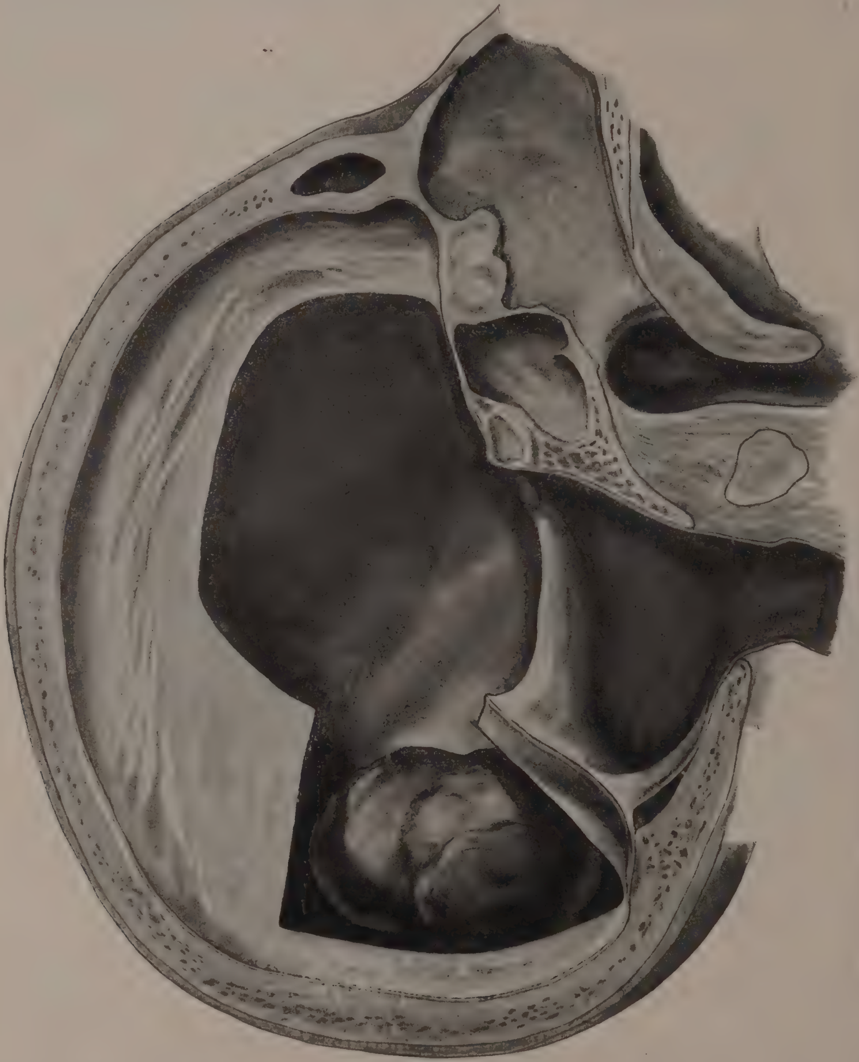


Fig. 237.—The extirpated tumor as in Fig. 236 put into a cranium with retained dural processes.

Such cortically situated, encapsulated tumors, can very well be taken out with the fingers, but only in parts of the brain, in which the resulting unavoidable increase in pressure and exertion of force will not cause too much damage. The entire convexity of the cerebrum may be mentioned here; of course, one will proceed with the greatest caution, by slowly feeling one's way. It was done thus in the case just mentioned.

Whereas the tumor had its broad base upon the dura, and its size could thereby be recognized at once by palpating the latter, the conditions are less clear in the operation, if the tumor clings to the dura mater only by a thin pedicle; the development in that case, also, starts from the inner dural surface. Thus, in the anterior portion of the fossa Sylvii I observed a tumor as large as an apple, which was connected with the inner surface of the dura by a pedicle only as thick as a pea, but from its histological character, must have had its origin there. It extended to the region of the insula, and was successfully removed from the depth of the fossa Sylvii, whereupon a cure resulted.

However serviceable enucleation with the finger may be in suitable cases, it can scarcely be tried in the vicinity of the medulla oblongata, that is, especially not in the tumors of the cerebello-pontine angle (acusticus neuromata) (cf. Fig. 239). The danger of exerting a dangerous, even directly fatal pressure upon the vital nuclei on the floor of the fourth ventricle by the insertion of the finger is too great.

Tumors of the Substance of the Brain

The method of extirpation of the real brain tumors varies, according to their kind and condition, their situation and accessibility, according to whether very vital parts or less sensitive parts of the brain are affected. On the other hand, it is of comparatively little operative significance, from what point in the substance of the brain the tumor originally grew, whether it was from the cortex inwards or from the depths within outwards. Important as these relations are symptomatically, and much as in giving a clinical judgment we must strive besides diagnosis of tumor, to recognize the manner of its growth and extension, still all this plays a very slight rôle in the technic. It remains the same, whether after opening of the dura mater, we see the new formation on the cortex immediately before us, or if we must seek it in the depths.

The *cortically located tumors of the substance of the brain* can be noted upon the exposed and usually non-pulsating dura, by their color which shines through, and by a certain prominence; rarely and only when it has considerable consistency, does palpation give useful results, since the tense dura blots out fine distinctions. After separation of the dura, they can mostly be recognized by their color which differs from that of their normal surroundings; only the diffuse infiltrating gliomata may form an exception. In my experience, the tumors that go out from the brain substance, do not become adherent to the inner surface of the dura, so that the dura need not be removed. If the favorable form, the encapsulated tumors, is concerned, the tumors can be enucleated in the same way as those growing out from the cerebral membranes into the substance of the brain.

Far less favorable for extirpation are the cerebral tumors that are not encapsulated. With them, nothing remains after ligation of all the larger afferent vessels, but extirpation with knife and scissors. If the tumor varies noticeably in consistency from the mass of the brain about it, as may be the case in sarcoma and even in glio-sarcoma, the removal of the new formation resembles an enucleative process and one may all the more safely count upon complete extirpation, if the surrounding cerebral substance is softened to a greater or less degree.

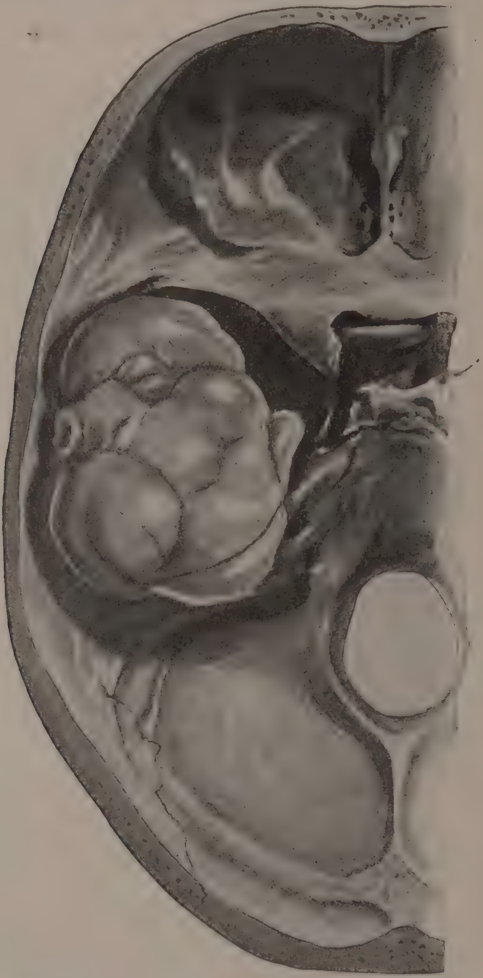


FIG. 238.—Cranial basis from above with the tumor placed back in position.

But unquestionably, the least favorable forms are the infiltrating gliomata; in them, even after the anatomic incision into the brain, one can not always say with certainty where the tumor stops and the normal tissue begins; how could this be possible in an operation in which all sorts of difficulties are present? One must cut as far as possible into the sound substance with the knife or the half sharp spatula, and will frequently enough leave behind parts of the new growth.

The difficulties just mentioned, are felt in increased degree, if the tumor has developed exclusively in the medullary layer. When the dural flap has been turned aside, the flattened convolutions and the blotted out fissures, occasionally too a striking dryness and lack of lustre in the surface of the brain, prove that the clinically established symptoms of cerebral pressure were really evoked by a narrowing of space in the cranial cavity. For the recognition whether the tumor is really in the place expected and how deeply it has its seat, palpation with an exploratory needle (acidopeirastic), as well as exploratory puncture and withdrawal of little portions of the tissue are indispensable, since feeling with the forefingers has scarcely ever in my experience proved to be of any avail. Since in operations on the brain one must be prepared for all possible occurrences, a skilled assistant should be ready to investigate at once microscopically the particles that have been withdrawn. We must mention that the slide preparations made after the hardening

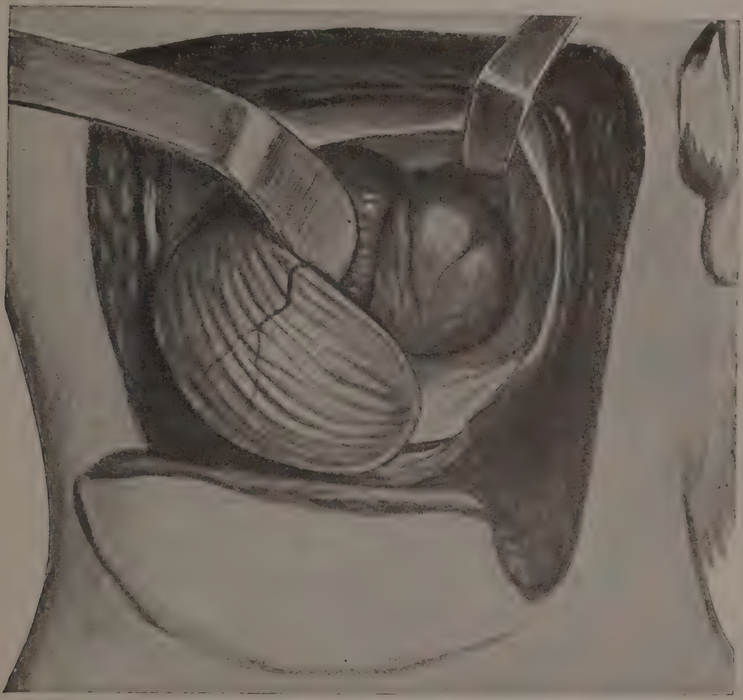


FIG. 239.—Extirpation of an acusticus tumor.

afford far safer and more certain results. Occasionally, one may decide as to the further procedure from the nature of the fluid withdrawn.

The acidopeirastic method was introduced in 1856 by *Middeldorf* and is serviceable in some cases; he tried it first in encephalocele, cephalhæmatoma and hydrocephalus. Far more important however, is

Cerebral Puncture

While this was formerly performed after the dura or the brain had been exposed, *Neisser* and *Pollack* deserve the credit of having systematically developed it in a way that it may be performed while the skull-cap is still intact. The clinical experiences reported by these authors have afforded us manifold explanations, which could be reached in no other way. Their positive results are concerned, apart from the determination of hydrocephalus internus, with intra-cranial hemorrhages and their residues (hæmatiodin), to the contents of cysts, purulent and seropurulent fluid, furthermore, to withdrawn pathological tissue (necrotic parts of the brain and particles of tumor), finally to bloody pial edema, in syphilitic leptomeningitis. At other times, the negative findings led to a change in the diagnosis which had formerly on the basis of clinical symptoms been made either as abscess or tumor of the brain.

According to my experience, the puncture in general is easily performed in the entire region of the calvaria, since the covering of soft parts is comparatively thin; at any rate, a strongly developed muscle in the temporal region may be a hindrance in so far as the canula can not without difficulty reach the opening bored into the bone. But in puncture of the cerebellum, in a young woman with a short very fat neck, I found that on account of the thick layers of fat, the hole that had been bored into the bone could not be found with the hollow needle in spite of all my efforts. Since we did not wish to dispense with the result of the puncture, nothing remained but to chloroform the patient and after lengthwise incision and pushing aside the periosteum, to bore through the bone with a fraise and then to make a puncture through the dura which was now exposed to view.

The danger of hemorrhage must be considered. The branches of the A. meningea media, in general also the cerebral arteries and the large sinus can be easily avoided by the canula, since their course is comparatively regular. But whoever has rather frequently laid bare the surface of the brain knows that the course and the size of the pial veins can not be calculated at all; they are often large in accessory fissures, thin in the central fissure. They have, moreover, hard walls and are easily injured. When the brain is sound and pressure conditions are normal, they possess it is true, a slighter lumen; but if brain pressure be increased, they may dilate considerably, and their injury is then certainly of importance. Even the finest canulas would then not be safe. I have myself seen in cases of tumors of the brain, the dural veins attain even the thickness of a heavy lead pencil. Finally, arteries and veins are so far misplaced by growing tumors, that every anatomic scheme may prove erroneous.

Concerning the danger of infection, it can be avoided with certainty from the outside, but not from purulent foci within the cranial cavity. Thus, in a young man, in whom *H. Oppenheim* diagnosed a tumor in the parietal region, I found a solitary tubercle, which in two places, had been transformed into abscesses (cf. Fig. 240). *Neisser's* puncture would have opened the purulent foci, which, covered by a very thin layer of cerebral cortex, were found right below the free arachnoidal space, which was not shut off by adhesions. Besides, the pus, as the operation showed, was under very high pressure, so that in the puncture, that was performed after exposure of the cortex of the brain, the fluid oozed forth beside the canula. The same would have happened in puncture through the cranial walls even had they been left quite

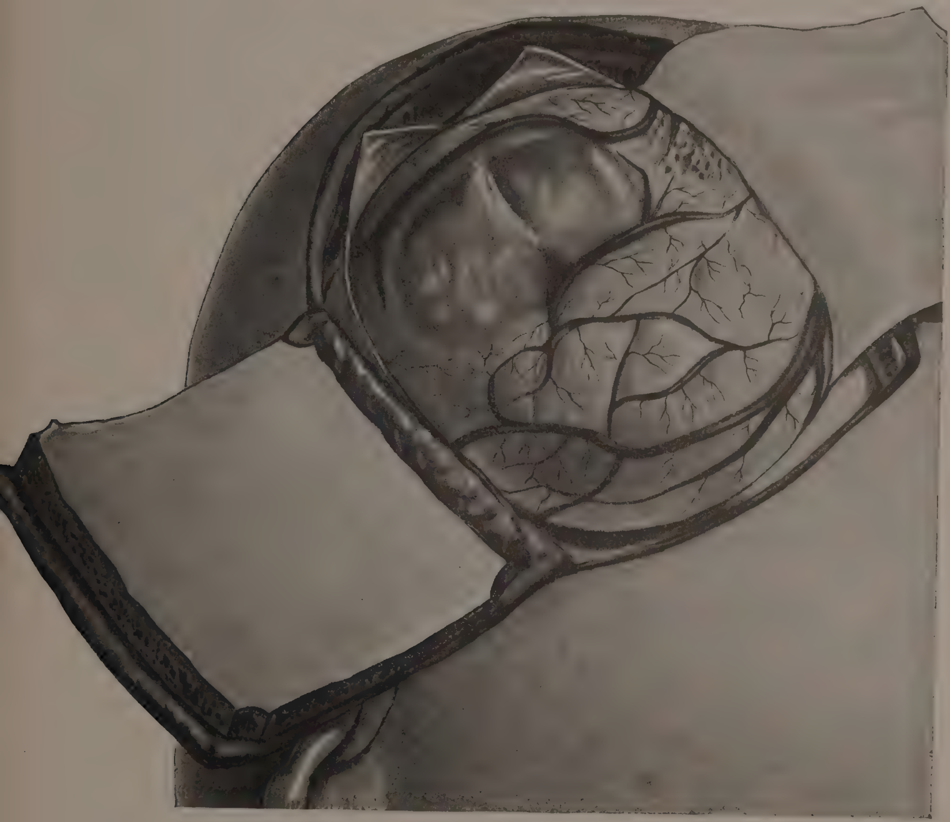


Fig. 240.—Solitary tubercle in the parietal lobe, transformed into an abscess.

intact. This pus, it is true, was not septic, contained no staphylo- nor strepto-cocci, but it was so full of tubercle bacilli that it might have been declared a pure culture. It could not be regarded as indifferent if considerable masses of such infectious germs should reach the arachnoidal tissue. The one protection against this dangerous occurrence would have been the immediate performance of trepanation.

But I have seen still other unpleasant phenomena appear after Neisser's cerebral puncture, which to enter upon in detail here, would carry us too far afield. However, one must admit that it represents a perfection in our methods of investigation. But it should only be used when all other methods of cerebral diagnostics have already been employed. Occasionally, too, we have reached important conclusions as to the seat and the extent of the disease, also as to the histological character of a tumor. In order to meet from the first the possibility of all dangers, cerebral puncture should be performed only, as I always do it, when all preparations for the trepanation have been made, in order that in case it should be necessary, it too may be performed without the slightest delay.

Opening of Cerebral Abscesses

When there is suspicion of cerebral abscess, I scarcely ever perform exploratory punctures through the dura. For from the puncture opening in the brain which often, because of the thick and tough pus, can not be made quite narrow, if the dura mater is intact, the pus may penetrate into the arachnoidal spaces and here cause infections. As a rule, I cut the dura mater also when opening the cerebral abscess in flap form and this with the base below; in this way, one gets the best view and approach to the cavity of the abscess; at the same time one can wipe off every drop of pus that oozes forth from the puncture canal of the brain.

Whereas in the operations for epilepsy and tumors, we have before us an aseptic area, we must, in case of abscess, protect the arachnoidal mesh from all contact with the infectious pus—whether it be of septic or tuberculous nature. We attain our goal most certainly by *protective tamponade of the subdural space.* This is accomplished by inserting strips of sterilized vioform- or iodoform gauze between the cut edges or the dura mater and the surface of the brain throughout the entire operative area. Only after exactly carrying out this occluding tamponade, do I proceed to the puncture and the opening of the abscess. The latter may be undertaken at once, if it is a question of abscesses lying quite upon the surface, which are recognized immediately after opening the dura. The layer to be severed then usually no longer consists of functioning cerebral tissue. These were the conditions in the case portrayed in Fig. 240.

But if we have to open abscesses lying in the depths of the medullary layers, our first task is to establish accurately the seat of the purulent focus. First, I puncture the exposed brain with a bare canula, and for this purpose I employ one with a large lumen up to 2 mm. in diameter. If the pus in the abscess is under pressure and viscid in consistency, it oozes forth from the inserted hollow needle without need of suction. But if no pus appears, I apply artificial suction, at the same time drawing out the canula slowly. If the result is negative, one must puncture at various depths and in several directions. The pus may be so thick and viscous that it does not even follow suction. Then in case of well founded suspicion of abscess, one should plunge the knife unhesitatingly through the substance of the brain down into the abscess, in order to see the pus emanate through this broader canal; it is the only way of saving the life that is threatened.

If the abscess has been found by the puncture, the canula is kept in its place, then one runs a scalpel along it, cutting through the layer of the brain which covers the purulent cavity, corresponding to the size and the depth of the abscess, a distance of several centimeters; or instead of the knife one uses the dressing forceps and by opening it thrusts aside the masses of the brain. The edges of the wound are held apart by elevators that are rectangularly shaped and blunt, the pus is carefully sponged out and the entire cavity accurately inspected for none too rarely, one perceives new pus oozing forth from a small place in the depths, and the probe leads to a second cavity. Then it is most to the purpose to follow the probe with the little finger and to divide the intervening wall bluntly, but cautiously and yet sufficiently, so that finally we have to deal with a single cavity in order to create as favorable conditions as possible for recovery. One must remember, furthermore, whenever opening a cerebral abscess, that several of them may be present, without any communication with one another. When the cavity has been freed from pus, in the acute septic abscess, one should not scratch off the infiltrated walls, because new infections might thereby be caused. If a real abscess membrane is formed, as happens in chronic as well as in tubercular abscess, the membrane should be removed. I place in the deepest part of the cavity, one or two rather good-sized drains according to its size, and tamponade round about these with vioform- or iodoform gauze.

Cerebral Prolapse

After aseptic brain operations, the primary wound should be sutured if possible, because a great disadvantage in keeping the seat of trepanation open, and in tamponade, is prolapsus cerebri. Even when there is no increase in brain pressure, when the wound is perfectly aseptic, and the pia mater uninjured, in a few days—though only exceptionally under such circumstances—the brain may protrude in such a manner that secondary suture causes difficulties. If one must, as in suppurative processes, continue the tamponade for some time, the protrusion of the brain especially if the pia was removed to a considerable extent, usually takes on huge proportions, as is shown in Fig. 241. It concerns the same case portrayed in Fig. 242. In the 28-year-old patient, the larger purulent tubercular focus was with difficulty dissected out with unopened scissors from the normal substance and the tamponade accomplished with iodoform gauze. The picture was drawn by the artist fourteen days later during a change of bandages, while the head was open and the occipital lobes exposed. The very soft prolapse of the brain had attained a circumference of about one and a half times a man's fist, and hung over the rear edge of the wound, like a bladder filled with fluid; below, it formed large folds like those of a double chin. From the seat of excision in the brain, which scarcely extended below the upper niveau of the prolapse, clear cerebro-spinal fluid kept dripping drop by drop. The lateral ventricle had certainly not been opened during the operation.

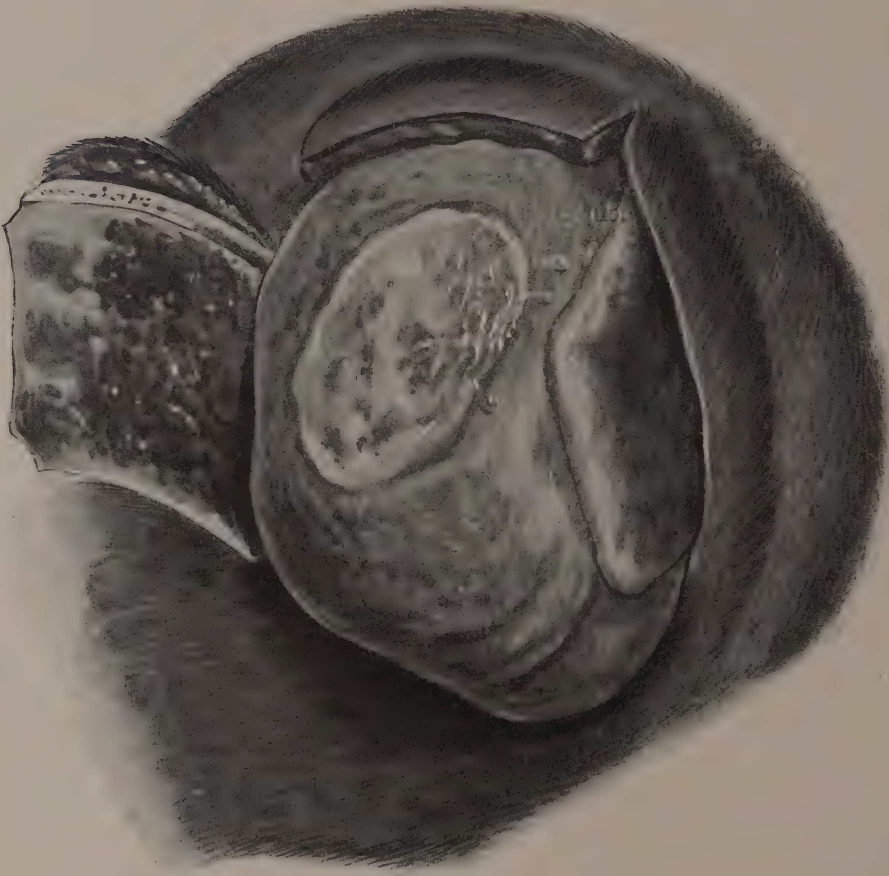


FIG. 241.—Postoperative prolapse of the brain. (Compare Fig. 240 and the text.)

Eighteen days later, the good general condition as well as the condition of the wound permitted it to be closed plastically. Under chloroform, the skin of the vicinity which had shrunken, was so far loosened that it could by sutures be drawn up to the skin of the superimposed trepanation flap, naturally under severe tension; small gaps remained uncovered which in the direction of the sagittal wound area reached a finger's breadth. Five weeks after the plastic operation the wound was completely healed, the place of trepanation protruded only a little (cf. Fig. 242).

I attempt to cover such prolapses of the brain by the enveloping skin. As the process was successful in that exceptionally difficult case, it succeeds all the more easily in less extensive prolapses. To deal with smaller prolapses, a moderate but lasting compression is sufficient. This is accomplished in suitable cases by means of strips of adhesive plaster beneath which are some layers of sterilized gauze. If the wound is aseptic, and there is no retention or new suppuration in its depths I have seen such prolapses even after attaining the size of hens' eggs, with the assistance of occasional treatment by caustics, become covered with an integument, even without any plastic operation—sometimes, however, with a certain protrusion which disappears in the further course, as the contraction of the scar increases. I do not approve of removal of the part of the brain that has prolapsed. Still one must confess that for the larger part they consist of edematously

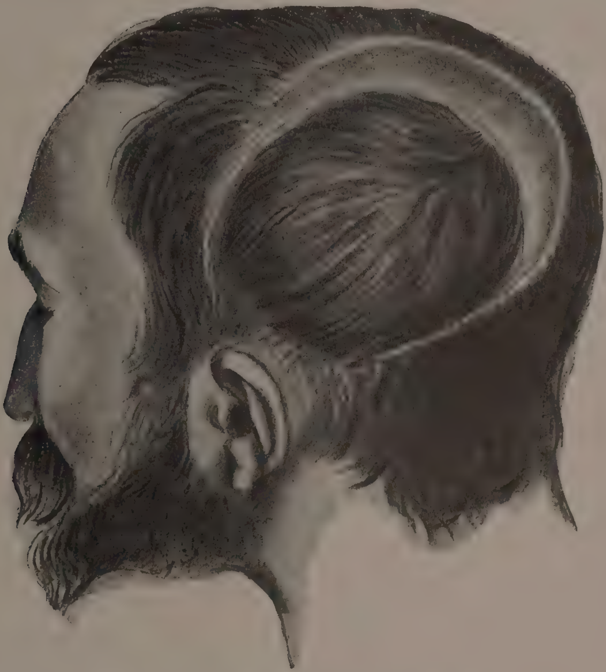


FIG. 242.—(Compare Fig. 240 and the text.)

soaked and inflammatorily infiltrated tissue, that is not pure brain substance, and that not very much danger exists in cutting off the smaller prolapses. But the prolapse usually forms very soon again after excision, unless sutures have been at once accurately drawn over the place. One may, however, for special reasons, be forced to cut off the prolapse.

The conditions are different and far more difficult when the prolapse of the brain occurs with simultaneous increase of the internal brain pressure, whether this increase in pressure be evoked by tumors, hydrocephalus, hemorrhages, abscesses, inflammatory processes, or by other causes. Thus in many cases of extensive cranial opening, especially if the tumor the presence of which was assumed has not been found, or could not be removed, the brain protrudes so considerably out of the gap, even during the operation, that it seems as if it could not again find place in the cranial cavity, and yet this may be attained after raising the upper part of the patient's body and turning back the probably much too small dural flap by sewing up the skin under the greatest possible tension; in this way, the entire wound may gradually be closed.

But if with continuous increase of pressure in the cranial cavity, the trepanation gap must be kept open by tamponade, there appear in as short a time as possible severe prolapses, which can only be pushed back if the cause of the increase of pressure can be removed. In abscess, for instance this can easily be effected, as soon as, after its opening, the surrounding inflammatory infiltration retrogresses and the entire process shows a tendency to heal.

Decompressive Trepanation

In a broader sense, there belong to the cerebral prolapses also those protrusions of old trepanation places, which usually develop in recurrent tumors, or in cases in which the removal of the tumor proved to be impossible. They are covered according to the operative procedure, that had been followed, by skin and bone or only by skin, in the temporal and cerebellar region also by muscle, in more or less extended places always by large scars, and therefore may quite plausibly be designated as cerebral hernias, to distinguish them from the prolapses.

It frequently happens that associated with the increasing protrusion at the site of trepanation, the most painful symptoms of cerebral pressure decrease. These favorable effects have led us to create an artificial cerebral hernia, when there is a brain tumor that can not be localized, that is, to perform a so-called decompression trepanation. In order to attain a secure safety valve formation, one must make the osseous opening considerably larger than the osteoplastic flaps, by removing with the gouging forceps on all four sides of the opening, together with the periosteum, a strip, 1 to 2 cm. wide. The dura mater should also be opened freely.

In all such operations the pronounced protrusion of the brain which appears usually immediately after incision into the dura, is very troublesome; it may proceed so violently that the soft cerebral membranes and the corticalis are torn by the sharp edges of the dura and the bones, thereby resulting in severe functional disturbances, since such injuries cause hemorrhages into the cerebral substance with consequent edema, and thereby again a more severe prolapse. In order to practise the greatest caution possible, I use small blunt scissors to make the dural flap considerably smaller than the osseous breach, next make connective cuts from the four corners of the dural flap to the corners of the bony breach and then turn back the lower three of the dural flaps that have been formed over the edges of the cut bone, so that they are entirely covered.

The immediate danger in decompressive trepanation may be regarded as slight, and the operation is a beneficent method of alleviation, wherever in the brain the undiscoverable or irremovable tumor may have its seat. It appears as justifiable as, for instance, gastrostomy in cancer of the œsophagus, as colostomy in inoperable cancer of the intestines, as gastroenterostomy in inoperable carcinoma of the pylorus. Like these it, too, is able to increase the duration of life. Still more important is its favorable influence upon the headaches, vomiting and visual disturbance for the former reaches the highest degrees, while the perpetual vomiting very soon leads to exhaustion of the organism, and neither symptom can be combated in any other way. Choked disc also retrogresses, frequently even with striking rapidity; there is an improvement in the clearness of vision, and if the fibres of the optic nerve have not already suffered too severely, the patients are at least preserved from blindness till their death. This is all the more important since in some tumors, choked disc appears very early, when other severe disease phenomena have not yet developed. Finally spasms that may be present and also deliria retrogress, and the feeling of vertigo becomes less annoying. Horsley has pointed out that subacute encephalitis may evoke the phenomena of tumor of the brain; in such cases, decompressive trepanation has directly curative effects.

On the other hand, it is usually of little avail in those cases in which it is a matter of rapidly growing tumors, on or near the surface of the brain. The diffuse gliomata and gliosarcomata which embrace large areas of the cortex and the medullary layers are especially unfavorable in this respect. The "safety valve" formation here affords only brief alleviation to the condition of the patient; soon there is formed an excessive protrusion through the osteoplastic flap, if it has healed at all by the progressive growth; the suture is burst open or the fresh scar breaks down. Rapidly the neoplastic masses press forward from all sides and can by no means be held within bounds. Every removal, every scraping only incites the proliferations to increased growth. For such patients, doses of morphine are the only method of alleviating the physical pangs and also the psychic pangs that unfortunately are frequently present.

Puncture and Subcutaneous Continuous Drainage of the Ventricles of the Brain

For *puncture of the lateral ventricle, Th. Kocher* prefers, since his main section is to a certain extent in the sagittal plane, such places as coincide with this direction. According to his experience, the most favorable place for this puncture is above and in front of the bregma, where the sagittal and coronal sutures join. Two cm. or better, because of the sinus longitudinalis, three cm. from the middle line, he pierces downwards and backwards; at a depth of 5–6 cm. one reaches the ventricle distended with fluid. Kocher performed drainage also in this way; in his opinion the process is best insured against injuries of the opposite ventricular wall, if one inserts the tube only into the upper part of the cavity and prevents its further progress forwards by fixation. On the other hand, v. *Bergmann* punctured the lateral ventricle from the forehead, boring directly above and somewhat medianly from the tuberositas frontalis, through the cranium, and inserting a hollow needle towards the back with a slight inclination downwards and inwards so far into the brain that the fluid was emptied by gravity.

Where there is a morbid accumulation of fluid, the ventricular walls are more or less distended according to the amount; they may even become extraordinarily thin, so that the cerebrum finally resembles a water bladder with weak walls. In such unusual conditions, puncture in any part of the calvaria finds the fluid not very far below the surface. Even in moderate hydrocephalus internus I have found it convenient to puncture above the middle of the sinus transversus about 2 cm. above the protuberantia occipitalis externa, in the horizontal plane, straight forwards or somewhat obliquely upwards and forwards; then one reaches the posterior horn. Drainage of the lateral ventricle may also be well performed in this manner.

I have twice undertaken *puncture of the fourth ventricle*, but only after I had freely exposed the dura of both hemispheres of the cerebellum in its middle division together with the sinus occipitalis. Precisely in the angle of the inverted Y (⅄) formed at this place by the dural covering of the medulla oblongata and that of the cerebellum, I inserted the hollow needle in the sagittal plane forwards and downwards, about 45 degrees above the horizontal line, slowly and carefully, until the fluid flowed off. In this way, it is true, the top of the fourth ventricle is pierced, but the vital nuclei on its floor remain uninjured. I saw no bad results follow either of these punctures; on the contrary, they exerted a favorable influence upon the severe phenomena of brain pressure.

The treatment of hydrocephalus has up to this time shown very unsatisfactory results. Open drainage especially of the cerebral ventricles is too dangerous, for with the continuous wetting of the bandages, decomposition and infection can not be permanently avoided. It is true that by using the greatest caution, I have avoided these dangers for several weeks, as was proved by repeated bacteriological examination of the clear fluid withdrawn but I have never succeeded in effecting a cure.

For continuous withdrawal of the increased amount of liquid I use, like *Mikulicz*, a gilded silver tube with thin walls, just 2 mm. in diameter (to be obtained from *Windler*). A small flap containing only skin and galea aponeurotica with a base of about 2 cm. is dissected loose above the place that has been chosen in the cranium, and the periosteum is split lengthwise and turned aside. The cranium is perforated with a drill at least 2 mm. in diameter, the dura is not injured. Now the hollow needle enclosed within the silver canula is inserted through the dura mater so far into the brain in the direction of the lateral ventricle or the posterior horn, that fluid flows off. While the little tube is held fast in place, one draws out the hollow needle, shoves in, in its stead, a little rod of hard steel of the same size (knitting needle) and with the sharp bone forceps cuts a groove completely around the canula about 1 cm. above the surface of the bones, so that after the removal of the steel rod, the canula can be broken off at this point. Now with a pair of narrow pointed scissors, it is slit lengthwise on two opposite sides, down to the skull, and the two silver prongs thus acquired are turned outwards at right angles, until they lie upon the surface of the bones. They serve to hold the little tube to a certain extent riding upon the bone, so that it can not slide down farther into the depths. In order to prevent its slipping, the periosteum loosened on both sides of the drilled hole, is laid over the bent prongs, and so fastened by a few catgut sutures that the lumen remains free. Through pressure upon the canula, or by drawing it out slightly, one may cause the fluid to flow off freely and then begin the fastening. The canula is cleaned from whatever blood may be present by inserting a sound into it, and then the flap of skin covering the opening of the canula is carefully sutured. The little tube ends in the subcutaneous tissue, and nowhere touches the line of sutures. When the little tube no longer draws off fluid, it must be removed; for this, simple incision into the skin suffices.

At first, until solid adhesion takes place, the ventricular fluid drips from the wound and the puncture canal, soon, however, into the subcutaneous tissue, forms an edema, and is absorbed. If one has undertaken drainage of the lateral ventricle, in the region of the temples or above it, one sees the lids on the same side or of both eyes, swell edematously, and within a few days, the swelling go down again. The process repeats itself as a sign that fluid is flowing off. Even when upon decrease in the size of the ventricle the inner end of the canula is misplaced by cerebral substance, fluid may trickle out between the silver tube and the cerebral canal. Precisely this slow off-flow is desirable.

This process comes into question also in those acute accumulations of fluid which in inflammatory processes of the membranes of the brain, for instance tuberculous meningitis, meningitis cerebro-spinalis, appear in the ventricles. W. Schulz has seen at least transitory benefits from ventricular punctures in epidemic cerebro-spinal meningitis.

In ossified cranial suture, that is in a completely rigid cranial cap, one should add to continuous drainage, in order to remove more rapidly the increased intra-cranial pressure, a valve formation without opening of the dura, for the dura adapts itself to the condition formed by the emptying of the ventricular fluid. These are quite different here from those in decompressive trepanation, performed because of inoperable cerebral tumors, in which according to my experience, without extensive opening of the dural sac, no result worth mentioning and above all, no lasting result can be attained, because with the advancing growth of the tumor, the tension of the dura and its firm resistance become greater and greater.

This safety valve formation together with ventricular evacuation is to be taken into consideration also in those cases in which with the existing cerebral tumor the accumulation of fluid in the cavities of the brain becomes so large as to signify immediate danger to life. Only some time after evacuation, when the threatening phenomena have been got rid of, will one proceed to extirpation under essentially more favorable conditions.

Epilepsy

The most important type for surgical treatment is *Jacksonian* epilepsy; it represents no disease sui generis, but a symptom-complex which appears in many diseases of the brain and its membranes, that is, it may be evoked by the most varying causes. First of all, we must separate the *traumatic cases*. Conditions are simplest if an injury to the cranium has affected the motor region, and a hemorrhage, a splintering of the bone, or a depression, the formation of cysts or scars, inflammatory or suppurative processes, directly affect the cortex of the brain. Such traumatic epilepsies have for a long time been treated operatively by cutting out scars of the soft and of the osseous skull cap, necessarily performing a trepanation, removing splinters of bones, emptying cysts and abscesses, cutting out also those membranes and parts of the brain that have been changed by the scars. There are cases enough in which the findings in the skull virtually forces the knife into the surgeon's hand.

In a second series of patients, *Jacksonian* epilepsy is evoked by *intoxications*, like lead poisoning, alcoholism, uræmia, or *infections* like pneumonia, meningitis. These forms belong to the surgical realm just as little as those that are developed on the basis of hysteria.

On the other hand, among functional neuroses, I must mention for the sake of completeness, the *reflex epilepsies*, which occasionally present the *Jacksonian* type. Occasionally, any scar on any part of the body may be a starting point for the convulsions. Those scars are especially feared that are adherent to the periosteum or nerves and are very sensitive to pressure. At times the patients perceive an aura, which emanates from such a scar, or pressure on it directly brings on the epileptic attack. The knowledge of such conditions is old, and *Dieffenbach* has performed excisions of scars in the soft parts, even amputation of limbs, in the hope that with the removal of the cause the disease also would disappear, a hope that was frequently enough disappointed, but since in these operations, it is not a matter of cerebral surgery, mere reference to them must suffice.

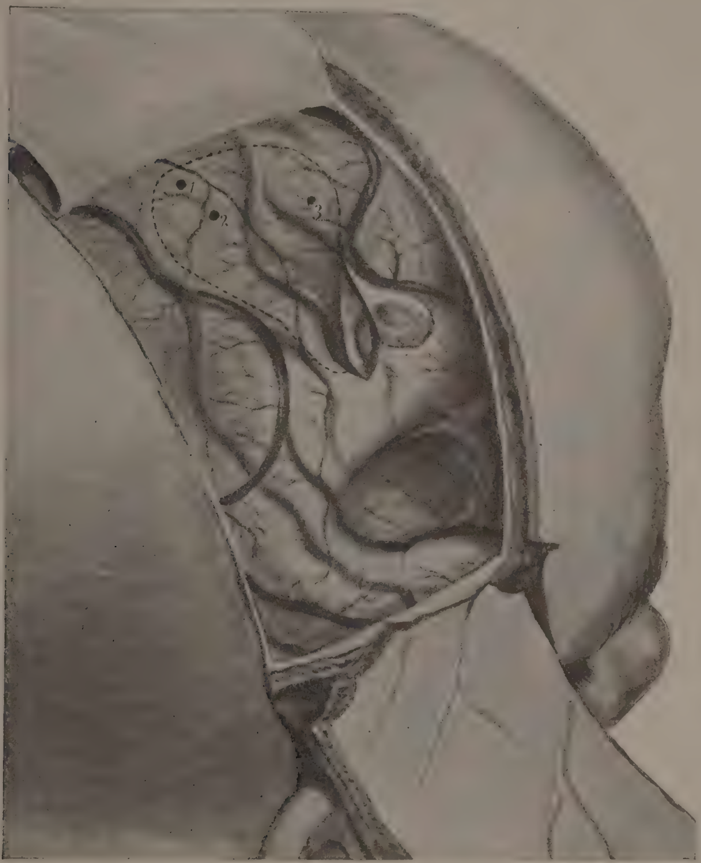


FIG. 243.—Opening of the central area in Jacksonian epilepsy because of infantile cerebral paralysis (encephalitis). Boy, 10 years old. Periosteal flap formed downward, dura flap toward the sinus longitudinalis. Large cortical and small subcortical cyst, atrophy of the cortex. Excision of the primary spasmodic center of the hand and the forearm, demarcated in the figure by a dotted line; 1, 2, 3, the foci of these parts of the extremities determined by faradization with one pole. Cure and disappearance of the idiocy.

Besides the traumatic epilepsies, those forms that attach themselves to *infantile cerebral paralysis*, come into question operatively. Here upon opening the dura, we usually find meningo-encephalitic focal diseases, extensive edema of the arachnoidea, also arachnoid cysts, finally scar formations. The chronic inflammatory overgrowths may transform arachnoidea, pia and corticalis into a single indivisible cicatricial layer. The edema of the arachnoidea must always be removed before we continue the operation; this is attained by superficial scarification or scraping with a small pointed knife, and it is best to undertake the little incision in the most dependent places of the operative field. The trickling of the subarachnoidal fluid which immediately results, is furthered through slight compression with pledget gauze, and soon the convolutions and fissures in the brain appear clearly. In scraping, one must be extremely careful to avoid the vessels; for if such a vessel, usually a vein, is pierced, extensive sugillation may arise, which with its reddish brown surface color, makes all orientation impossible.

Several times I have found subcortical cysts (cf. Fig. 243) which usually are of very small circumference, but in isolated cases contain even several hundred cubic centimeters.

In my opinion, the surface of the brain should always be exposed, once one has decided to operate. Only in this manner, is there any possibility of recognizing and removing any disease foci that may be present. The standpoint that when the dura looks and pulsates in normal fashion, mere trepanation should suffice, seems to me untenable. I have seen, when the dura was sound and had normal pulsation, severe changes in the brain after it was opened; at other times, such changes were unimportant, though pulsation of the dura was lacking or slight. If one finds such severe changes in the motor region, that they explain the symptom complex, our surgical interference is ended with the necessary treatment of this focus. In such cases one will feel best satisfied if operation be performed, for then one can hope that with removal of the cause the sequelæ will be gotten rid of also. Féré has already said in his work upon epilepsy that only such treatment as is directed against the cause of epilepsy may hope to be successful.

On the other hand, changes so easily recognized are by no means always present; at times, the visible disease products are too insignificant, as in some of our patients in whom a leptomeningitis in circumscribed extension was present. In all such cases, one must employ faradic stimulation preferably with one pole, determine the primary spasmodic center, puncture at this place in search of a subcortical process, and then if necessary make the incision. Only if no such process is found, is the excision of the cortical center concerned to be considered, according to the example of *V. Horsley*. The method had been taken up with enthusiasm in his time; the enthusiasm gradually waned, and surgeons finally gave up the procedure, as I think, unjustly. If one looks through the literature one can prove that the majority of surgeons establish the center according to anatomic landmarks, and this, in my opinion, is a mistake; therefore also all former operations in which this happened, must be subjected to a specially searching criticism. Even after a large opening in the cranium, one sees often enough the faradically determined center lie partly hidden under the bones, from which one must remove additional pieces. Just as little can one depend on the anatomic details of the exposed surface of the brain; the variety and variability of the fissures and the convolutions and of the pial veins are far too great.

Now when the center has been found by faradic stimulation, the excision follows in connection with the soft cerebral membranes, down to the white substance, that is, of an average depth of 5 to 8 mm.; the bleeding vessels are ligated in the usual fashion; if necessary, a little piece of sterilized gauze bandage is laid upon the defect and withdrawn before the wound is closed. The danger in the operation is not increased by a small excision of the cerebral cortex; the paralyses and sensory disturbances that may appear at first, retrogress on the whole.

Against this method it has been objected that the defect operated upon could only be cured by a scar, which again might cause epilepsy. Logical as these conclusions are, only practical experience can decide as to their validity or invalidity in man, and my experience teaches that the resulting scar by no means regularly has such an effect of exciting anew the disease.

But we may also find anatomic changes in a place in the brain other than that corresponding to the cortical field primarily stimulated. Then it must be left to the surgeon's judgment, whether he will content himself with removing the disease focus or whether he considers it necessary to cut out in addition that part of the cortex (cf. Fig. 243).

Concerning *general genuine epilepsy*, Kocher has proposed forming a movable valve. As is well known, he entertains the view that sudden increase in pressure in the cerebro-spinal fluid which is present in a great quantity and is already under high pressure, immediately produces loss of consciousness and evokes general convulsions. In some epileptics I have observed convulsions during the operation and have seen the brain at the same time press out of the trepanation opening like a dark bluish-red bladder stretched to the utmost. *Kocher's* valve formation has produced a number of unquestionable improvements, even some cures, so that in consideration of the futility of all other methods, and of the numerous otherwise hopeless cases, this method deserves all the more respect, since according to my experience, the operation is not dangerous.

I operate above the anterior central convolution, and moreover, in right-handed persons, on the right side, though I have observed in very rare cases, disturbances in the innervation of the opposite half of the body, which, however, passed away very rapidly. The osseous closure of the cranial opening should be prevented, but for this purpose the piece of bone that has been cut out need not be entirely removed; it suffices to take away from all sides bone substance to the breadth of a centimeter or a finger, as explained on page 1032. In order to complete decompression the dural flap should not be sutured down. Usually it has shrunk so by contraction, that when it is put back, it does not entirely fill out the defect; it is best to proceed in accordance with the directions on page 1032.

2. DISEASES OF THE SPINAL CORD AND ITS MEMBRANES

Surgical interference is concerned with all those paralyses of the spinal cord in which total or partial interruption in conductivity is caused through injuries or compressions of the cord. A pressure paralysis may be caused by every special restriction in the spinal canal, whether it is a matter of real tumors of the spinal cord and its membranes, or whether diseases of the spinal column of various kinds lead to secondary compression.

As to the

New Formations of the Membranes of the Spinal Cord

the great advance in the operative field rests, to a certain extent, upon the perfection attained in the treatment of wounds and in surgical technique, but we must ascribe an influence at least as great to the extraordinary advances in the diagnostic realm which the last decade has brought us, and moreover, this does not refer alone to diagnosis of tumors of the spinal cord, but especially to the certainty with which surgeons have succeeded in many cases, in determining the exact location of the tumor (*level or segment diagnosis*). There is needed in operative interference a knowledge of the vertebral arches beneath which the tumor is to be sought. The surgeon has only *one* thing to point the way for him, to guide him as he penetrates into the depths, namely the vertebral process accessible to his sense of touch alone. With recognition of this fact, the shortest way is mapped out for our operative attempts, the operation being the less injurious, the smaller the number of vertebral arches to be removed. We must know, therefore, the situation of the various segments of the spinal cord relative to the situation of the vertebræ concerned. The segments always lie higher than the vertebræ having the same number, and the difference becomes larger, the farther we go from above downwards; quite important individual fluctuations are, moreover, present, as *Reid* proved.

Now for the sake of clear vision, at least two vertebral arches—except, possibly, in the lumbar portion of the vertebral column—should be removed. But if the tumor has developed in the ligamentum denticulatum, one must, in order to be able to reach it at all, raise the spinal cord, after splitting the dura mater, and this can be done with the necessary caution, only after it has, to a large extent, been exposed. In cases here concerned, I always sacrifice the vertebral arch; if one attempts to retain the arches, one increases the difficulty of the operation without gaining any advantage. The supporting power of the spinal column does not suffer essentially, even from the loss of seven arches, an operation I performed successfully in the case of a 38-year-old patient; at least there is not so great an impairment as to warrant our

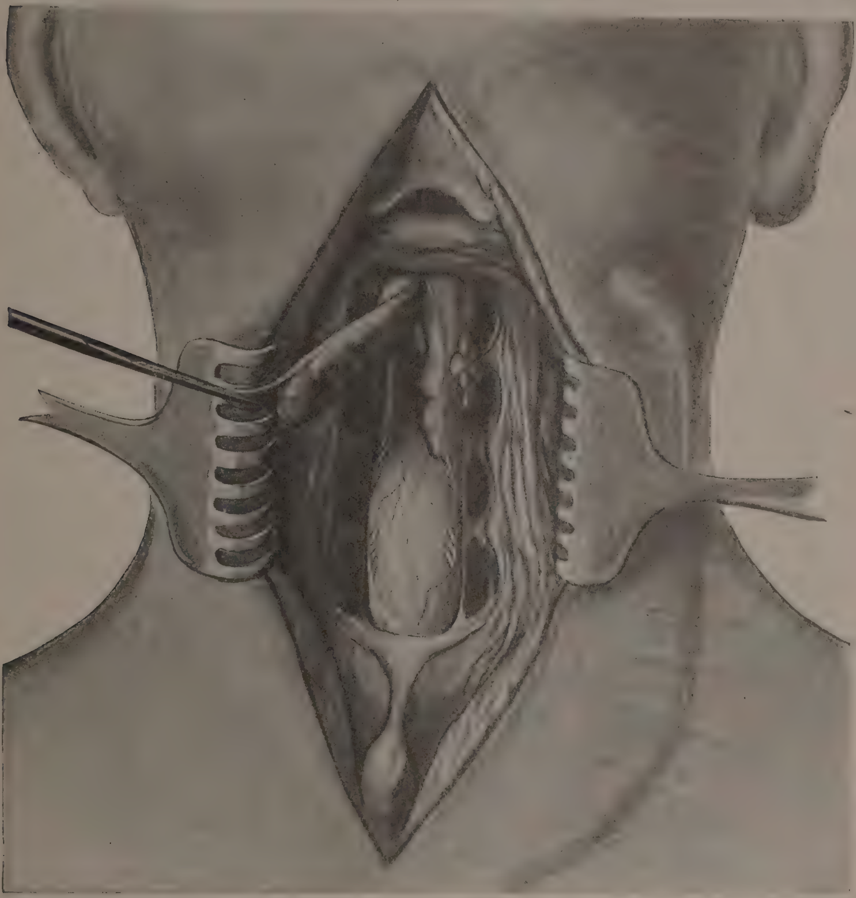


FIG. 244.—Man, 23 years of age, twice operated without success. 3 1/2 months later resection of the 2d to 5th cervical arc, removal of the fibrosarcoma of the dura mater reaching into the atlas, which grew toward the spinal cord and left there a deep cavity. Complete cure and retrogression of the severe paralyses.

making more difficult an operation which is in itself dangerous enough. When the spinal canal is widely opened, and the epidural fat has been bisected in the middle line and together with the venous plexus shoved aside bluntly, the dura mater spinalis is usually exposed to sight. If an extradural tumor is in question, it is already recognizable, and can, after the approach to the spinal canal is sufficiently widened, be removed. Thus I dug out from its bony bed an enchondroma proceeding from the sixth vertebral cervical segment with a sculptor's chisel. But if, as usually happens, we are dealing with an intradural new formation, the dura usually appears bluish and very tense, the pulsation not being at first visible.

But even when the dura appears normal, it must under all circumstances be opened, just as I do in the brain, unless there are present extradural changes that completely explain the disease picture. Three times I have had to operate upon patients into whom prominent surgeons, one, even twice in the same patient, had cut in vain, because they stopped at the dura; in two of these cases, I extirpated an intradural tumor, in the third case I found present the severe changes to be discussed below as chronic arachnitis. I must state that failure to open the dura is a technical mistake. If the dura exhibits decided tension, one can but rarely without opening it, conclude as to the presence or absence of an intradural process. Occasionally, one sees the intradural tumor through the dura, or feels it as a hard spot. Above and below the tumor—I have observed both—the dura may show pulsation.

Now, if one opens it with a knife, which must always be done lengthwise, clear cerebro-spinal fluid spurts forth in considerable quantity; by widening the incision with scissors, the fluid sometimes pours out in streams, at once filling and overflowing the large cavity of the wound, and after withdrawal by syringe, this process is repeated a second and a third time. 120 g. of fluid were caught by me in one case, and even then a large part of it was lost. When the flow has subsided, the dura mater is split lengthwise, through the entire length of the wound, and in favorable cases, the new formation at once appears, if it lies behind, then frequently only in a small area. A widening of the wound and the removal of one or more arches are frequently necessary.

If the tumor be situated laterally towards the ligamentum denticulatum I have found that quite on the edge of the cord, a long thin stripe is prominent by reason of its color, which verges on grayish red. By the insertion of a blunt tenaculum, the spinal cord must be raised a little from one side, then at that place the tumor, which had been tightly pinched between spinal cord and dura comes forth from the depths.

The arachnoidea frequently passes from the spinal cord without any boundary over to the tumor, surrounding it in a kind of capsule; when that has been cut into with the scissors, the new formation with the capsule can be easily removed from the cord with a blunt instrument. The dura must be removed with scissors, throughout the length in which it has intergrown with the tumor.

The new formation lies in a corresponding groove of the spinal cord, which here seems flattened, but after removal of the tumor take on a more and more circular form, although at this place it remains considerably thinner than its segments immediately above and below. It is astonishing how rapidly the deep hollows which represent the bed of the tumor, in some cases level up before our very eyes during the operation. If we get such a patient for autopsy, a few days after the operation scarcely a trace of even very deep cavities can be perceived. In like manner, the ability of the spinal cord to recuperate is astonishingly great, surpassing all expectation; twice in men of 23 and 28 years, who as a result of an intradural tumor in the cervical segment, had been virtually paralyzed in all four extremities, I witnessed the return of the essential functions within a few weeks.

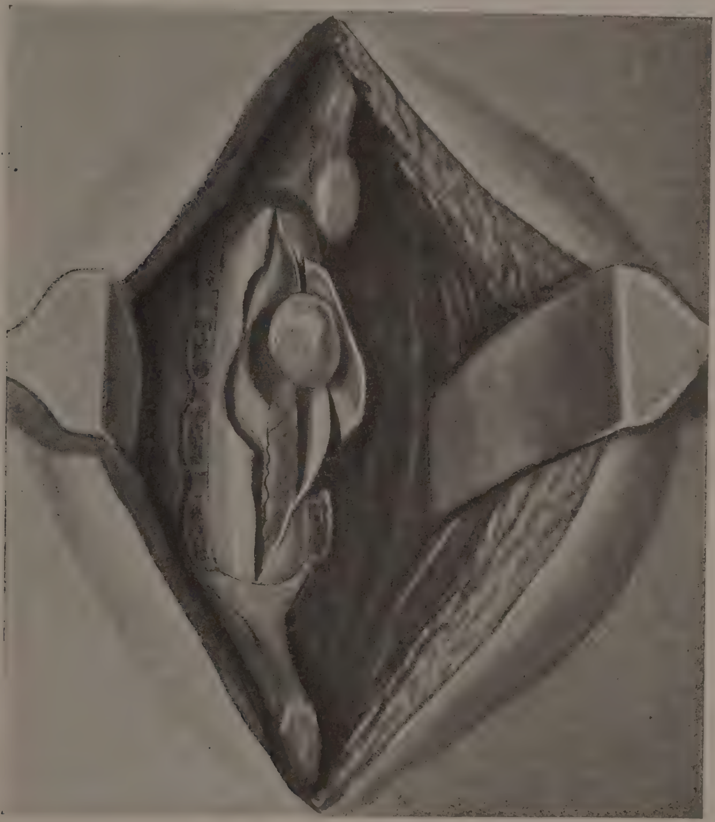


FIG. 245.—Psammoma of the dura mater in the dorsal part, removed in a 65-year-old patient. Cure after 8 1/2 years duration.

Operations on the spinal cord should, if possible, be performed in one stage. In 26 cases, I had to perform two stage operations only twice, once because of extraordinarily severe hemorrhage, the other time because I had to remove 7 dorsal arches, and the patient was too weak to undergo the severe operation all at once.

Especially unfavorable are cases of *intramedullary tumors or tumor-like growths;* the tumor may have affected so much of the spinal cord, or be so diffusely infiltrated, that its extirpation may be impossible but the smaller intramedullary tumors and those encapsulated are very well adapted for extirpation; twice have I made a lengthwise incision of 2 to 3 cm. exactly in the posterior commissure into the spinal cord, and once opened a cyst as large as a pea, the other time a softening focus as large as a bean—effecting a cure in both cases. In the same way, if I were certain as to the diagnosis, I should make an incision for an intramedullary new growth. In those cases it was a matter of severe spinal paralysis, with all the phenomena of the growth of intervertebral tumors, while the operation showed only the formation of indurations, comprising the dura as well as the archnoidea and pia as cause of the compression. The patients recovered from the very extensive operations in which I cut out all cicatricial masses; in one man I had to remove, in addition 7 dorsal arches. The paralyses, too, retrogressed to a considerable extent.

Isolated *tubercle formations* and also *gummata*, which evoke compression phenomena, and do not react to specific treatment, must be removed surgically. Since, as is well known, old gummatous processes of the bones, skin and tongue are most quickly cured by operations, so likewise, in the central nervous system, in the brain as well as in the spinal cord, one should not wait until the nerve elements are incurably destroyed by compression, but decide upon operation in time.

The real *tumors of the spinal column* present but rarely objects for operative treatment; they may be either primary or metastatic, in the latter case usually being carcinomata. In one case of cancer of the breast, in which only a few weeks after extirpation the first phenomena of compression of the spinal cord appeared, during the entire observation period no deformity of the spinal column nor the least painfulness could be found, but the diagnosis had to be made as follows: metastatic carcinoma probably of one vertebra with proliferation into the spinal canal, and compression of the spinal cord. Since all symptoms on the part of the spinal column were lacking, we could assume the exact location of the growth only from the nervous disturbances. The autopsy proved our assumption correct.

Finally *spondylitis tuberculosa* on account of kyphosis, but also because intervertebral granulations and suppuration compress the spinal cord, may give occasion for operation. However, one sees pareses of the extremities even of long duration improve under treatment by extension, disturbances of the bladder disappear, anomalies of sensibility and the reflexes compensated. These improvements are due to the relief of the spinal cord as a result of straightening the kyphotic curve in the vertebral column, resorption of intravertebral exudates, shrinking of the granulations here located; naturally all these factors may be working in unison. From these facts emanates the demand that in spondylitic paralyses one should wait for some time; also in older persons of 45 and more, I have succeeded repeatedly with orthopædic measures, and one will decide to operate only when these methods fail. In any case, the waiting should not be protracted too long, so that the inhibitions of conductivity in the spinal cord may not be aggravated into incurable breaks.

Now there are a number of cases in which the development of the disease as well as the existing phenomena point to a mass compressing the spinal cord, but in which laminectomy discloses as the only or at least the main cause of the important disturbances a locally circumscribed accumulation of cerebro-spinal fluid under very high pressure (*meningitis serosa spinalis*). In three cases, I found more or less extended indurations in the membranes of the dorsal part of the spinal cord. My operative experience has taught me that even in vertebral caries, the phenomena of compression of the spinal cord need not be conditioned only upon the kyphotic misplacements of the vertebræ, not alone upon the filling of the spinal canal with granulations and pus. The secondary congestion of fluid may also contribute its share in evoking spinal paralyses, or at least in bringing them into full being.

It is certainly very remarkable how such an accumulation of fluid with its resulting pressure can develop at a quite definitely localized place in the membranes of the spinal cord. For here in the normal state, the equilibrium of the fluid is very soon restored; but in pathological conditions, mechanical changes, whether it be misplacements, occlusions, or adhesions and overgrowths may be the cause of congestion of the fluid in a circumscribed area, say, for instance, within the limits of two or three vertebræ. It follows directly from this, that the cerebro-spinal fluid, is not secreted exclusively by the plexus chorioidei, as was formerly assumed, but in part at least also by the arachnoidea.

The affection of the arachnoidea leads not only to formation of adhesions, and to increased exudation, but has also the consequence that the resorption capacity of the arachnoidal tissue at that place is, if not entirely lost, at least considerably diminished (*arachnitis adhæsiva circumscripta*). The diagnosis can be cleared up neither by lumbar puncture nor by puncture at the place of compression.

Besides the chronic form of this spinal paralysis for which I have shown a perfectly analogous example from the cerebellum, there is also an acute or subacute form evoked by purulent necrotizing bone processes in the spinal canal. We have an analogue to the latter form in the disease called by aurists meningitis serosa cerebralis acuta, occurring in purulent processes of the middle ear and the neighboring bony parts, here in my opinion usually representing a propagated inflammatory edema, and able to exhibit all the phenomena of septic meningitis or abscess of the brain. Under these conditions the operation discloses suppuration in the cavum tympani, in the antrum and mastoid cells, probably also a perisinus extradural abscess. The temporal lobe of the brain shows severe tension of the dura and no sign of pulsation, but cerebral punctures nowhere disclose purulence but only the clear fluid, which being under strong pressure, even afterwards, streams out of the opening made by the puncture. After operative removal of all the diseased tissue, the severe cerebral phenomena disappear. In the same manner an acute congestion of fluid confined to a small space may be evoked in the spinal column and in the spinal cord by purulent changes in the vertebral bones, as I have observed; here too we may interpret the process as a propagated inflammatory edema.

Since in neither form—neither in the more acute nor in the chronic variety—does spontaneous retrogression occur according to my experience, the only possible treatment is laminectomy; at the same time then in non-purulent processes, the dura mater should be opened, and this is the more desirable inasmuch as we do not yet possess any means of distinguishing meningitis chronica spinalis ex arachnitide chronica, or whatever term one chooses to apply to this disease, from tumors of the spinal cord.

3. DISEASES OF THE PERIPHERAL NERVES

Tumors of the peripheral nerves may be either benign or malignant. In the former, usually fibromata, one need not undertake resection of the nerve trunk; it suffices to extract the new formation in such a manner that, after splitting the nerve-sheath lengthwise, one dissects out the sound branches, and places them to one side and severs only those that are inseparably connected with the tumor. This is what I did, for instance, in a neurofibroma of the tibialis, and cured a 23-year-old workingman, who suffered from the most violent radiating pains; the strikingly slight sensory and motor phenomena of functional loss which followed directly upon the operation, have totally disappeared. But even the benign neuromata may be inoperable, as a result of their multiple appearance, in which not separate trunks with their ramifications, but exceptionally even the entire peripheral system is attacked. We must place the neuroma plexiforme, which, as is well known, represents a manifestation of elephantiasis and must be extirpated like a solid tumor, with the multiple fibromata.

Because of the pains which are often intense, amputation neuromata are feared; they usually arise when the severed nerve ends become intergrown with the scar. My teacher, *Richard von Volkmann*, avoided this technical error by drawing out and sufficiently resecting the nerves.

But in the nerves there occur also, though rarely, new formations, which belong to the softest, medullary forms of sarcoma, and myxosarcoma, evince a decidedly malignant nature, break down, ulcerate, and creep on centripetally, within the neurilemma and the perineurium, so that for instance, a nodule arising first in the forefinger and proliferating into the vola manus (palm) may lead to a series of amputations and finally to the futile exarticulatio humeri; after death, which soon supervenes, the nerve trunks are found to be diseased, all the way to the spinal canal, and covered with tumors. These forms of neuroma may proceed even from the smaller nerve trunks, so that the nervous origin may always be overlooked unless a special dissection of the parts is made directly for this purpose. In these tumors also, a new formation of nerve fibres and that too of the medullated fibres, takes place.

Because of the imminent danger of recurrence, the mere extraction of such a tumor which is centrally located in the nerves must by no means be thought of. At most, in those forms, that are located more firmly upon the nerves laterally, one may content one's self with removing with the tumor only that part of the nerve fibres that are immediately adjacent to the tumor; even this procedure often seems hazardous. Resection of the nerve at a considerable distance from the tumor, is far safer, but even this does not always prevent the formation of new tumors. One should undertake a nerve suture or neuroplasty after the resection, if the case seems to be adapted to it.

Simple extirpation, however, is suited only for tumors, which, still surrounded by an evident capsule of connective tissue, are in no way intergrown with the neighboring parts. If the latter has taken place, in most cases amputation and that at some distance from the new formation, should be considered, but even this does not always cure. Before one proceeds to it, one should, through a diagnostic incision, become acquainted with the relation of the tumor to its vicinity, and especially with the condition of the large nerve trunks above the tumor. Here one must watch carefully whether the nerve shows any traces of disease (reddening, small grayish red or red nodules) in order to determine the place of amputation according to the result of the findings.

If we are dealing with *injuries to one nerve*, through which its connection is lost altogether or in great part, nerve suture must be resorted to. If the injured ends can not be directly joined because pieces have been torn from the nerve, or which have to be resected because of crushing, we use little nerve flaps which we form from one or both stumps to restore the continuity; or we place a little tube of decalcified, that is, resorbable bone between both stumps, so that they may grow to meet each other in the path constructed for them. If this procedure is impossible also, we insert the restored peripheral nerve end in a neighboring trunk, after dividing its nerve sheath laterally. According to more recent experience, the regeneration proceeds more safely and rapidly if one implants the central stump also farther up in the same nerve trunk, so that we have a double connection. But in any case patience is necessary; it takes many months, occasionally a year or more, before the sutured or implanted nerve again exhibits functions in its peripheral division.

If peripheral nerves are imbedded in scars or in callus, and thereby hindered in their conductivity, they must be carefully dissected, and in order that the injuries may not develop again, so imbedded in normal elastic tissue that they become immovable.

Nerve grafting is of importance. If for instance, the facialis be permanently paralyzed, whether it be a result of total division, of severe cold, or of caries of the petrous bone, a peripherally restored end is implanted in the hypoglossus, or, though this is not so good, in the accessorius. The implantation may be made laterally; the process is safer if one divides transversely the primary nerve, and then joins its central piece with the peripheral part of the facialis. I had to wait 15 months after union of the hypoglossus and facialis, end to end, until the first slight twitchings appeared in the eyelids of a young girl; then there followed gradually a very satisfactory restoration of the activity of the facialis, which owing to cold had been entirely lost for 5 years.

Such favorable results have led in infantile spinal paralyses, in which transplantations of tendons and muscles frequently have excellent results, to the attainment of the same goal by means of neuroplasty. This train of thought rests on absolutely physiological foundations and, it is hoped, will prove to be practical. Tibialis and peroneus are here above all concerned. A few remarks about the transplantations of muscles may be to the point here. It was Nicoladoni, who first acted upon the idea of using in incurable muscular paralysis a neighboring sound muscle and its tendon, in order to compensate the disturbance evoked by that paralysis. This method has opened numerous fields to operative orthopædics. We treat today in accordance with the above principle, not only paralytic deformities of the lower extremities, but also paralyses in the arm and the hands, especially radialis paralysis, furthermore, even the spastic contractures, like those in *Little's* disease, and finally arthrogenous contractures, such as appear in or after chronic inflammations of the knee joint.

O. Förster had the fortunate idea to improve the severe spastic paraplegias of the legs, by resection of some of the posterior roots of the spinal cord, which is performed after wide opening of the dural sac, it being quite indifferent whether their paraplegias are to be traced back to a spinal or to a cortical disease. *Tietze* effected some remarkable results with the new method. Naturally this serious operation is to be used only in severe cases, which can be helped in no other way.

Of *spasms in the realm of the peripheral nerves*, mainly those of the facialis and accessorius are treated surgically, the former by stretching the nerves, or by resection of the peripheral trigeminus branches which excite the cramp. It seems to me justifiable in severe tic convulsif that can be influenced in no other way, if the symptoms demand it, to transversely divide the facialis, and to implant its peripheral end in the hypoglossus in the manner described above. This might be a physiological method of effecting a permanent cure, since such severe spasms are dependent on central conditions. The irritated facialis nuclei would then be excluded and replaced by the normally functioning hypoglossus nuclei. Transverse division through the hypoglossus evoked no subjective disturbances in the above mentioned young girl, though it did, of course, produce a certain atrophy of the half of the tongue concerned.

The accessorius spasms may be of the severest degrees and affect the vicinity to a large extent. They also evoke violent pains in the contracting muscles. In a 64-year-old woman whose picture is reproduced here (Fig. 246), a slight accessorius spasm with twitching in the head had appeared 20 years previously; gradually as a consequence of great misfortunes and intense excitement, the very violent and extremely painful spasms have spread to the entire musculature of the body down to the abdominal muscles, so that virtually only the lower limbs remained unaffected. In lighter cases, resection of the accessory nerve, which must be undertaken in great extent, affords alleviation, even cure; in severe cases, one must resect the affected muscles (sternocleido-mastoideus and the musculature of the nape); but in the most

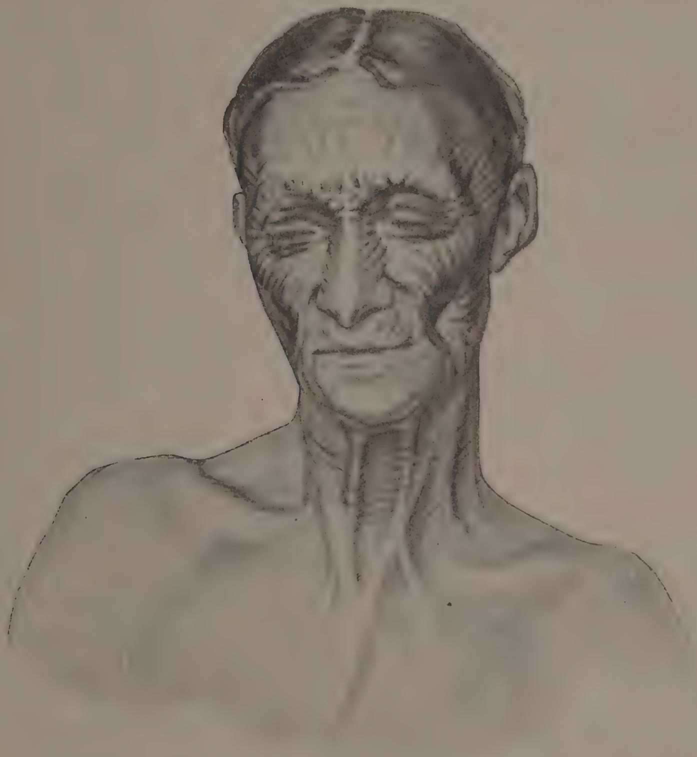


Fig. 246.—Spasm of the accessorius with extended affection of the vicinity.

severe cases, one does not always even by such extensive measures secure a favorable result.

Of the *neuralgias of the peripheral nerves*, mainly those of the trigeminal and the occipital nerves come into consideration for operative treatment. It was Schleich who first undertook to evade an operation by injection of his well-known anæsthetic mixture in the vicinity of the affected nerve trunk or, in the case of a larger nerve like the sciatic, directly into it. *Schlösser* uses 70 to 80% alcohol, and injects it directly, for instance into the diseased branch of the trigeminal, with the conscious purpose of destroying its ability to function and to kill it; the nerve branch concerned should, except for the neurilemma, be brought to degeneration and resorption in all its parts. The purpose of this method is, therefore, to replace nerve resection.

On the other hand, *Lange* employs large quantities of physiologically isotonic fluids; in the sciatic for instance 70 to 150 c.c.; in the first and second trigeminal branch, 30 to 50 c.c.; in order to inject them under stronger pressure into the sheath and its direct vicinity; he wishes to effect thereby a loosening, stretching or mechanical tearing of the nerve fibres. W. Alexander has gained good results in sciatica by injecting Schleich's solutions not into the nerves but into the painful muscle insertions in the trochanter major and tuber ischii.

Now, when sensibly executed general treatment or such injection cures have been proved useless in severe neuralgias, *operation* is to be considered. We must emphasize here that it should not be considered as the very last remedy, as unfortunately happens but too frequently, and only be attempted when all other methods have failed. Unquestionably, as a result of this view, many neuralgias which in the beginning could have been cured by insignificant peripheral operations, are aggravated by their long duration. For even if microscopic examinations of the resected peripheral trigeminal branches have usually shown no remarkable results, still there must be certain changes in the nerves, which cause the condition of irritation, and thereby the neuralgia. While these changes are, in many cases, at first peripherally located, they progress centripetally in the course of time, and finally in such deep-rooted cases, peripheral operation can no longer be of any permanent value.

In trigeminal neuralgias two operative methods are concerned, differing in respect to the difficulties of execution as well as in their danger: first, those undertaken outside the cranial cavity, extracranial operations; secondly, those in which the cranium is opened, intracranial operations.

One will be all the more likely to see results follow upon extracranial nerve operations, if the cause of the neuralgia can be proved to be in the area of the peripheral ramifications, or if the pains are confined to one or a few end branches. If in the course of peripheral ramifications some disease foci are found (scars, tumors, etc.) by which the neuralgia might be caused, they must be removed at once. Here it is a question, usually not of nerve operations in the real sense, but it will be well to remove to a larger extent branches that are exposed in the wound.

Peripheral nerve operations have in a not inconsiderable number of cases led to permanent cures; more frequently, however, the results have been transitory, removal of the pains only for months or a few years. But even then the operation is a blessing for the patients; for in this horrible disease, every day free from pain is an immeasurable gain, and this gain is won with a gradually diminishing stake of danger and sacrifices. Theoretical statements are less to the point here than otherwhere. The best proof is afforded by the not insignificant number of patients, who have again and again presented themselves to the knife of the surgeon to be for a time at least relieved from their pain. Every busy surgeon has had such experience, and W. W. Keen in Philadelphia has reported a case in which a dentist had allowed himself to be operated upon 14 times in 13 years, for a trigeminal neuralgia. Furthermore, the late recurrences of the affection after peripheral operations have often proved far milder than the original disease, so that the patients were satisfied with their condition, and made no request to be operated upon again.

Mere total division of the nerves concerned is by all means to be rejected, since we know from various observations in man, and innumerable repeated experiments on animals, that, as a rule, a very rapid reunion takes place. But since not even extensive resection of nerves is finally satisfactory, I have been extirpating for many years the peripheral trigeminal branches in as large an extent as possible, that is, from the base of the skull to the finest ramifications, which can very well be done with the help of *Thiersch-Wietzel's* twisting out process.

In the most severe cases of trigeminal neuralgia, only extirpation of the ganglion Gasseri affords permanent cure; I have never seen a recurrence after this operation, and since my first operation—I have since performed 64—sixteen years have passed away. All my cured patients are happy, that at the expense of slight disturbances, they are freed from their horrible torture.

The severe occipital neuralgias also demand surgical treatment.

In very violent intercostal neuralgias, which mocked at all treatment, total division of the posterior roots has been carried out several times.

XIII

THE DIAGNOSIS AND TREATMENT OF NEURASTHENIA, PSYCHASTHENIA, HYSTERIA, AND BORDERLAND MENTAL STATES

BY

CHARLES W. BURR (Philadelphia)

NEURASTHENIA, PSYCHASTHENIA, HYSTERIA

Introductory.—My purpose is not to write exhaustively, because most of the subjects treated in this chapter have been thoroughly discussed by other contributors, but simply to emphasize certain points in diagnosis and treatment and to view from a somewhat different angle the question as to the relation of these different disorders to each other. First I must explain that the rather figurative and clumsy phrase "borderland mental states," commonly abbreviated to "borderland cases," is used to designate conditions of mental disease, other than hysteria, not serious enough, or not sufficiently typical, to be classed with the insanities and yet so severe as to make the sufferer's life abnormal, cause grief and trouble to his family, often, but not always, mental pain to himself and mayhap make him useless, even harmful, to the commonwealth. It includes all those persons who either show from childhood, or later in life develop, ideas and thoughts which give them a distorted view of things resulting in producing a miserable existence without causing absolute insanity. "Mental" means only that the symptoms are psychical and does not imply an immaterial, psychic, causation. It does not connote a psychogenetic cause of the disorder. Neurasthenia, hysteria and borderland mental states are all closely allied. They all have certain factors in common. Indeed it is possible that they are but different manifestations of a common trouble, the differences being dependent on variations of the personalities affected. They make up quite a large part of the clientele of every general practitioner of medicine. Correct and thorough knowledge of them is of great importance to the general practitoner because the sufferers always go to him first and only later, if at all, to the specialist, and because if properly treated and managed at the beginning, for management is the most important element in the handling of them, oftentimes years of illness can be avoided and even a cure brought about. Further, since many of them show signs of their presence as early as puberty or during adolescence the family doctor can do much to prevent their development, can make them abort instead of going on to fruition. In all, heredity is the great cause, i. e., they are manifestations of some congenital defect, caused by ancestral taint, which affects the entire personality, and if we were close enough observers and knew enough about the development of children we probably could foretell by puberty what would be the fate of any given boy or girl as far as susceptibility to any of these conditions is concerned. Certain occurrences in childhood are danger signals of later trouble, but they are not infallible signs. Thus stammering, frequent sleep-walking, night terrors and day terrors, both of which are sometimes epileptic equivalents, obsessions in childhood, bed wetting, and cyclic vomiting are all evidences of nervous instability which may or may not be precursors of serious hysteria, neurasthenia, etc. Idiopathic epilepsy beginning in childhood usually leads to other and different results. Saint Vitus dance, so far as my personal experience goes, has been an infrequent event in the lives of people who later on developed so-called functional nervous or mental disease. This is rather surprising and it would be a valuable thing to study the matter statistically on a large scale. True insanity in infancy or childhood ends fatally or leaves the victim an imbecile. Rarely, even little children may pass through an attack of acute mania, which is about the only insanity children are subject to, and apparently recover entirely, but the outlook for permanent cure in such cases is always bad. There is always recurrence and early mental breakdown. Whatever significance neuroses occurring in childhood may have as to the future health it is certain that the physician who has seen a child grow up, who knows its personality, knows and appreciates at their true value symptoms occurring in the adolescent or adult, which may mislead a doctor unfamiliar with the patient. Furthermore, though inheritance is the great cause of temperament and character and physical strength or weakness, yet environment has a great influence for both good and evil and often the family doctor can more or less control and thereby, by putting the child in wise and wholesome surroundings, occasionally prevent the development of neurasthenia, hysteria, or borderland states.

It is a trite truism to say that diagnosis is of great importance but it can not be reiterated too often. The first question in any case of disease manifesting itself through the nervous system is, is the disease functional or organic, or rather grossly organic, that is, does it produce lesions visible to the naked eye or by the microscope, or is it one of those affections which cause no visible mark or sign of their presence? There is probably no really functional disease, that is, no disease unaccompanied by either anatomical or chemical change in the cells of the organ or organs the functions of which have ceased or become perverted. Probably the diseases we call functional are in many, if not in all, instances due to chemical disturbances not sufficient to produce anatomical changes visible by our means of examination and arise not in the organs which show the symptoms manifest to us but in others apparently normal. For example, many nervous diseases have their origin in distant parts of the body, the brain being acted upon secondarily. Thus cretinism shows mental symptoms, but is a disease of the thyroid gland. Nevertheless, the classification of diseases into organic and functional is useful. It is particularly useful prognostically because, barring the occasionally successful surgical removal of a tumor, no chronic organic disease of the brain or spinal cord is curable save in so far as we are able to affect a symptomatic cure of certain syphilitic disorders, while many functional troubles are curable. Diagnosis is important in purely mental affections because it is necessary in every case to determine whether the illness is going to develop into a true insanity or whether it is really a borderland case which, though it may never be entirely cured, yet can be held in check or more or less improved. Failure to make a diagnosis early may result in total failure in treatment.

Neurasthenia.—At one time, indeed for a long period, this diagnosis was made with great frequency. Every woman who showed symptoms of too easy exhaustion, especially if she were nervous, irritable, emotional, slept badly and complained of numerous aches and pains, was either dismissed as having nothing the matter with her, save intense selfishness and laziness, or was dubbed a neurasthenic and then, with most doctors, an end. With advancing knowledge, with more care in study, the number of cases diagnosed neurasthenia has decreased, without any increase in the number of malingerers, until now some physicians even deny that such a thing exists and claim that in every case there is some accidently acquired cause, external in origin, some other disease, which is the real cause of the neurasthenia and relief of which will bring cure. Another view is that neurasthenia is really a psychasthenia, has not a physical basis, but is purely mental in origin, and needing only mental treatment.

All neurasthenia is not secondary nor is it purely mental in origin. There is a pure primary neurasthenia, not caused by external influences, not secondary to some other disease and not due to any mental stress, but dependent upon an inherent weakness in the person, a condition which, though it commonly first appears in adolescence or early maturity, yet depends upon congenital or hereditary causes. Primary neurasthenia is rare but it really exists.

The real anatomic seat and nature of neurasthenia is entirely unknown but it is allowable, for that very reason, to indulge in a little theorizing. That it is congenital, i. e., potentially present from conception, and that its real cause is a bad heredity, is surely true. We have no statistics on the matter; it is impossible to get them, but every physician who has had large experience in treating these people soon learns to look for and find signs of abnormality in the father or mother or both, or some or several of the near kin. The family weakness may show itself in many different ways. There may be some definite chronic disease or only a lack of power of resistance to stress and strain. The histories of the early lives of the patients always reveal much that is abnormal and they are strikingly similar. The physician soon learns to put to one side stress and strain, worry and trouble and grief, as anything more than mere exciting causes, because he finds that the disease occurs in their absence. Indeed he finds that it is not usually the people who are subjected to great stress and strain who break, but those who have never had any great weight to carry or no heavier than the average person carries without injury. Often he finds it occurs in those who shift all stress and strain to others and that often patients are of such a temperament that they do not realize great stress and strain because they have no sense of responsibility. He concludes that it is not a sequel of acute disease because he finds that neurasthenics are largely immune, for some unknown reason, to many of the acute infectious fevers and when attacked by them bear them well, and he finds that the secondary neurasthenia following such fevers in ordinary people, when it really is a neurasthenia, is usually quite rapidly recovered from. He finds, notwithstanding the popular notion to the contrary, that it is not a disease confined to the so-called upper classes, the so-called idle and lazy, but attacks alike the idle and the industrious, the playing class and the working class. For these reasons we are forced to look to the person himself, or more usually herself, to find the cause. Some writers have tried to explain it on the theory that there is no such thing as primary neurasthenia, that all cases are secondary and due to some existing but unnoticed or disregarded organic disease. Some, for example, maintain that in women it is always caused by disease of the genitalia, and point to the fact that ovaries and uterus are often undeveloped. It is true they often, indeed usually, are undeveloped, but this is only a sign of general poor development rather than the cause of the condition. The failure of development is only one sign of many of the anatomic defects which do not produce symptoms but are themselves the result of congenital, inherited defect. Undoubtedly acquired pelvic disease, not mere lack of development, causes many neurasthenic symptoms and ought always to be corrected when possible, but this is not the same thing as saying that neurasthenia has for its cause and explanation pelvic disease. There still exist some disciples of the school of reflex causes. Papers are still written to prove that eyestrain, floating kidney, spinal curvature, gastroptosis, unerupted or misplaced teeth are each and every one the one and only cause of neurasthenia, but it occurs in the absence of all of them and all of them may occur without the sufferer being at all neurasthenic. They are evils and ought to be corrected, but their correction will not cure primary neurasthenia. The real significance of the so-called reflex causes of neurasthenia is that they indicate undue irritability to stimuli in the person.

Of late years, and with the increase in our knowledge of physiological chemistry, much thought and time has been given to the study of the influence of disorders of the ductless glands in causing neurasthenia. Though some progress has been made in the physiology and pathology of these organs, our ignorance is still much greater than our knowledge, so that we are not on sure and known ground. It is very possible that the future will show that primary neurasthenia is due to congenital disturbance in the functions of some one or more of the ductless glands brought about by inherited causes. However, for the time being, and as a working hypothesis, Gowers' hypothesis of abiotrophy will serve to explain neurasthenia.

Gowers, some years ago, put forth the idea that some diseases might be due to an inherent weakness of the protoplasm of the cells of the organ affected. The cells, according to the hypothesis, are potentially strong enough to do only a certain amount of work and when that work is done, no matter how young the person may be in years, they grow old and either cease to work or work pervertedly. Now neurasthenia behaves just that way. The patient acts as if certain of his cells had done all the work they can do. There is no way to prove the truth of this hypothesis, but it explains, in some degree if not completely, what otherwise is a mystery, namely, that some people without any stress or strain, or disease or accident break down unduly and far too soon.

I shall describe the clinical history of neurasthenia very briefly. The great symptom is weakness, physical and mental—a coming on of great fatigue after slight effort, which persists even though all stress and strain, so far as such is humanly possible, has been removed. Along with weakness there is the irritability of exhaustion—light hurts, sounds, even music, cause pain. The stomach, indeed the whole alimentary system, works poorly, interruptedly, pervertedly. The cardiac apparatus is functionally upset. To think for any length of time is impossible. The sensory areas at the nape of the neck become the seat, not so much of pain as, of queer sensations which alarm the patient. Emotional balance is lost and, in a mild way, the moral sense becomes unbalanced as is shown by selfishness and perversity. Neurasthenia does not lead to crime in the patient but it often tries the temper of relatives. To give in detail the clinical history of neurasthenia would require many pages because the symptoms are so numerous, affect almost every organ of the body, and vary with the individual temperamental differences in the patients. The great symptom is, as said above, fatigue with irritability, not only emotional, conscious irritability but general irritability of all or almost all organs. The next important thing is the age of onset. Primary neurasthenia is a disease of the young. Your true neurasthenic begins his career of invalidism in youth or early maturity. Not infrequently we learn from the history that the patient developed normally till puberty but that soon after, or during early adolescence, a change was noticed in his or her temperament and character. The boy or girl, who previously seemed to have been entirely normal, began to show signs of muscular, digestive, and mental weakness. By mental weakness I do not mean feeble mindedness, far from it; often they are brilliant, but they lose zest in mental work and easily tire mentally. Emotionally they become moody and prone to periods of depression. In the same way, boys lose zest for muscular play and instead of wanting to eat too much as every healthy boy should they become finicky about food. At this early period it is impossible to foretell the future because many youths, boys and girls, pass through a period of unstable balance and emerge healthy men and women; others do not recover completely but improve and relapse repeatedly, and others begin at this early age a life of more or less serious invalidism. It is from the last two that the ranks of the neurasthenic, the hysteric, and many other types of the degenerate are recruited.

The onset of primary neurasthenia is always slow, so slow that for a long time no one recognizes illness. The relatives think of the patient as delicate, not very strong but very sensitive, and do not dream of sending for a physician, but regard the symptoms as a sort of visitation of providence, to be borne as best one may. Relatives of another type do not believe the patient is ill, but think she is lazy and pretending and selfish and that she should be compelled to do things. Sooner or later something happens within the patient, what it is we do not know, but serious symptoms come to the surface, and only then is it realized that disease is present. A sudden onset of illness always means an external cause and that the neurasthenia is secondary. Slow onset does not prove that the case is primary, however. The course in primary cases is as a rule like this: the patient complains more and more of tire and weariness, then she (it is much more frequently women than men who suffer) begins to sleep badly. She at first goes to sleep easily enough but soon wakes and is restless the rest of the night. Later sleep is long in coming, quick in departing, and leaves no sense of rest behind but rather weariness. Some patients dream much, others not at all. Appetite is lost or becomes capricious or variable. The patient often craves food she previously never cared for, usually indigestible and useless things which yet she digests well. There is discomfort after eating, a feeling of weight, or oppression or distention. The bowels become constipated or alternately constipated and loose. Pain and queer sensations are felt in the head. Pain may occur in any part of the body but the curious nape or occipital ache is characteristic. There are uncomfortable sensations in the cardiac region; the heart stops, or beats too rapidly, or seems to jump and turn over and there is a subjective feeling of dyspnea. There is always emotional depression. At the beginning it is not lasting, indeed none of the symptoms are. The patient gets better and worse, but there is a pretty steady trend downward. She may be excited and irritated but it is always the excitement of depression, or unhappiness. She feels her relatives do not treat her properly, do not appreciate her at her true worth. She thinks a husband, who may really be a loving slave, is a brute. This never becomes severe enough to become a delusion of persecution. There is a loss of mental power. She can not think for any length of time or on any subject requiring real thought. She may, and indeed often does, think perfectly well at the start, but brain tire comes on very rapidly. There are never any true hallucinations or delusions. What she wants to do she can do; what she does not want to do she can not do. This looks like wilfulness; really she exerts all the will power she can in doing what she wants and collapses when effort is over. Friends look on and marvel at the things she is able to do, then wonder why she can not continue, and think that she could if she only would, altogether forgetting that a spurt of effort is different from continuous exertion. The weakness increases until finally she is bed ridden, emaciated from lack of assimilation, depressed almost to melancholia, fearful of the future and sure she is incurably ill.

Traumatic neurasthenia is worth consideration by itself. It might be thought after what I have said against stress and strain, and external conditions as the cause of neurasthenia that I should deny its existence. I do not, because I am quite sure that stress and strain and shock of all kinds are exciting causes. My only contention is that the real cause, the predisposing one, is inherent and congenital. If a normal man or woman suffers an accident he or she does not become neurasthenic, but only the person predisposed. The symptoms are precisely the same as in neurasthenia from any cause. It is possible that the shock of accident causes chemical changes in the bodily functions.

Sexual neurasthenia in youths is a distinct type and is much more common in boys than girls. It occurs in late adolescence and early maturity, and most often in high strung boys of strong sexual feelings who have masturbated instead of seeking other means of sexual relief, and who later on, because of their high moral sense, have become conscience stricken or frightened by reading quack literature. The main symptom is fear of impotence. On their first visit to the doctor these patients do not speak of the things that really are troubling them, but recite a whole list of trifling symptoms for which no boy would bother to seek relief. When they get to know the physician they complain of all sorts of queer sensations in the genitalia. They have headache, say that they are thick-headed and can not think and complain much of vertigo, by which word they mean blurred vision. They have backache and a whole host of nondescript symptoms, and are much frightened by the occurrence of the acne of adolescence. Usually they have read a large amount of quack literature and are much frightened. Often there is fear that they are congenitally perverted, while the real pervert is not often perturbed by his abnormal emotions. It is necessary in examining any youthful neurasthenic to discover the existence of a sexual element because such cases require a work cure rather than a rest cure.

In addition to this true sexual neurasthenia of youth, women at the time of the menopause often develop many nervous symptoms and sometimes even a melancholia the exciting cause of which is occasioned by sexual irritability.

In spinsters and widows there is sometimes at the change of life a sudden development of sexual feeling demanding relief. It is a thing entirely outside the will, is often fought against, but sometimes leads to masturbation especially in the pre- or post-dormitium. The nastiness of masturbation horrifies these women and they hesitate to talk frankly to their physician. They present many symptoms of nervous irritability and may even become melancholic. When a physician suspects such a condition it is wisest for him once and for all to have a frank talk with the patient about the matter and then, having quieted her fears and assured her that her symptoms are only transitory, lay down a routine of life for her including exercise, work, interests outside herself, and for a time a mild sedative.

Diagnosis.—The first thing in making a diagnosis of neurasthenia is to exclude all other disease. Of course every sick person, no matter what his disease, should be thoroughly examined but especially should the examination be thorough in suspected neurasthenia, whereas in truth some of us neglect the supposed neurasthenic woefully and after a very superficial examination jump to a conclusion which often turns out erroneous. More people suffering from organic disease are assumed to be mere neurasthenics than the reverse. It is theoretically possible that anyone, no matter how strong he may be, may break if the strain be sufficient, but neurasthenia beginning in middle life is usually, in fact almost always, secondary and due to cardio-vascular-renal trouble. It is alleged that arterial disease itself may be caused by stress and strain but, since it occurs with equal frequency in men and women who have and have not been subjected to stress, proof is rather difficult. The middle-aged man, previously healthy, who begins to be conscious of his præcordium, who gets a little short winded going up stairs, whose sleep is somewhat broken, who cuts down his food because he finds eating makes him uncomfortable, who can not think as quickly as before, who is not sure of his judgment, is not, as a rule, neurasthenic: usually his heart muscle and kidneys and arteries need to be watched, for in them lies the trouble. Another disease to be excluded is syphilis. When there are clear symptoms of focal disease of the cerebro-spinal axis this is easy enough, but syphilis without the existence, at the time, of any other symptom may cause a profound neurasthenia. If there is the slightest reason to suppose in any case that syphilis may be present, a Wassermann test should be made and if a positive reaction is present antispecific treatment instituted. If the Wassermann test is negative but clinical signs are present, still treatment should be given. Syphilitic neurasthenia is very common after the secondaries have ceased and before the tertiary symptoms appear. A careful examination of the habits of patients should be made not only as to alcohol and other drugs but as to the sexual life. All neurasthenia is not due, as some writers would have us believe, to unconscious, repressed sexual desire but some of it, even in married people, is due to indulgence in unwholesome habits or to mere excess. The abdominal organs should be carefully examined. Trouble with the gall bladder and the gall ducts may give no local symptoms, or rather such slight ones that the patient pays no attention to them, and yet produce quite marked neurasthenia. Chronic appendicitis is frequent in neurasthenics, especially men, but it is probable that the appendicular inflammation is only part of a widespread colitis. Operation, however, often relieves the symptoms. Disease of the ovaries and uterus in women is not provocative of as much neurasthenia as some writers believe, but the generative organs should be examined and, if they need treatment apart from neurasthenia, they should be treated. To remove ovaries and uterus for some trifling local trouble will not only not cure the patient but lead to ills she had been spared till the removal. The blood should be examined for malarial parasites if the patient lives or has lived, no matter how long ago, in a region where malaria occurs. In those parts of the country where the hook worm exists the stools should be examined, because nothing else causes so pronounced a secondary neurasthenia as it. A hemoglobin examination and corpuscle count should always be made and no judgment formed from the apparent good color of the skin and lips, etc., because a quite marked anæmia may be present and yet the patient look full blooded. The existence of a pronounced anæmia of any type disproves primary neurasthenia, but a slight chlorotic anæmia is often present. The urine should be examined, not once, but repeatedly, because it is especially in the cases, in elderly people, in which there is only now and again a trace of albumin and occasional casts, but a constant low specific gravity, that the neurasthenia of Bright's occurs. When there are active signs of inflammation of the kidneys, mistakes are unusual because a different picture of disease occurs than neurasthenia gives.

Neurasthenia and melancholia are sometimes mistaken for each other. The essential symptom of melancholia is intense causeless emotional depression; physical weakness only comes later or not at all. True melancholia never begins till middle life. It never commences in adolescence or early maturity, though there are types of insanity in the young in which depression is a transient symptom, but it is soon replaced by much more serious symptoms. In severe melancholia there is always a profound feeling of self depreciation and self accusation. Such delusions never occur as a part of neurasthenia. If a patient who seems to be a neurasthenic begins to accuse herself of sin you may be sure you are dealing with melancholia. The diagnosis is important because the melancholic may commit suicide and therefore should never be left alone. Furthermore, an absolute rest cure is contraindicated in melancholia. The patient may need rest if physical weakness exists, but not the complete rest cure.

Hypochondriasis is sometimes mistaken for neurasthenia with disastrous results because the hypochondriac needs an active out-door life. He should be driven to physical labor and forced out of bed rather than be sent to it.

Hypochondriasis is a pure mental disease. Its great symptom is belief in the existence of some disease which disables. In the mild cases there is a delusional state, but not a definite delusion. The sufferer believes he is ill but does not know what his disease is. In the more serious cases he has a clear cut delusion concerning some one or several diseases. He has cancer or heart disease or syphilis or some other definite thing. He is not simply an ignorant man who, feeling sick, ignorantly makes a diagnosis. Such a man is sane and can be reassured. The hypochondriac is insane and has all the certainty of belief in the correctness of his own judgment, which the egotism of insanity causes and hence can not be reassured, can not be reasoned with. He does not present any of the earmarks of the neurasthenic. He does not show their manifest weakness and the irritability of exhaustion.

In the early stages adolescent insanity may be mistaken for neurasthenia but the picture taken as a whole is different. The great symptoms of adolescent insanity, dementia, catatonic spasm, and stupor, negativism, mutism, refusal of food, personal filthiness as to the bladder and rectum, wild excitement and delusions do not occur in neurasthenia. The presence of any one of them proves insanity.

Paresis sometimes shows only neurasthenic symptoms for several months after the onset or rather the neurasthenic symptoms are so marked and manifest that we neglect to look for minor signs of organic disease. Often they would be found if sought for. A little slurring or scanning in speech, slight tremor of the lips on speaking and in the tongue when protruded, some inequality of the knee jerks, some pupillary disturbance, one or another or several of these signs are almost always present. In paresis there is always a true loss of mental judgment from the very beginning, not mere mental tire but mental weakness. The paretic usually shows some silliness in manner and in his attitude toward life. He never realizes there is any mental disorder; he may realize physical weakness. The neurasthenic often fears insanity. Age also is important. It can not be repeated too often that primary neurasthenia never begins in middle life. Paresis usually begins at about 35 or 40 years.

Treatment.—In serious cases no other method of treatment equals the rest cure, for which the world is in debt to Dr. S. Weir Mitchell. How rigorously it should be carried out depends upon the severity of the illness. I will assume that we are dealing with a severe case, the patient having been ill for a long time and not having been helped by the other measures usually recommended. The four fundamental factors in treatment are: bed rest, milk diet, isolation, massage and electricity. Almost as important is the personality of the doctor. He must know neurasthenia and must superintenpersonally every step of the treatment. It is not enough for him to prescribe a system of treatment and let it work itself out; he must, by frequent visits and careful attention, see that it is carried out. On the other hand too friendly an attitude may, in the patient's emotional state, lead to evil. The physician must be master and in complete control, but he must be physician only and not a social friend. His psychological influence over the patient must be strong, but it must be confined to medical matters.

Isolation.—Every neurasthenic is better off away from her family and a serious case can not be treated successfully in the patient's own home. If the expense is not beyond the patient's means a private room in a rest house properly equipped or in a special hospital is the best place to carry on treatment. For those who can not afford such expense a bed in a ward of a hospital for nervous diseases is far better than trying to carry on treatment at home. For people in moderate circumstances a bed in a hospital ward is preferable to a private room, because they are relieved from worrying about the cost and paradoxical as it may seem, seeing other nervous patients does not injure them. Sometimes it has a beneficial influence. There are many reasons why separation from home and family is wise. Neurasthenics are always emotionally unstable. They are prone to be peevish, fretful, selfish, and depressed. When at home it is much harder for them to retain self control. They crave sympathy and make great demands on those whom, when they are well, they care most for and are most thoughtful about. Married women, when neurasthenic, are for many reasons much helped by being in greater or less degree separated from their husbands. Sometimes husbands do not know how to treat their wives and hence do much harm. It is best that the sexual emotion should be kept in abeyance. Mothers are often the worst companions for their daughters. The parents of neurasthenics are often themselves abnormal and create a very evil atmosphere around their children even when they think they are sacrificing themselves for their children's good. They often undo everything the physician is trying, by mental means, to do. Apart from the separation from the family it is well to keep the patient from having visitors. Friends always know some easy way to cure; they are usually obsessed with the idea that what the patient needs is diversion and they often preach. Such preaching does harm. How extreme the isolation must be varies greatly. In some cases it is best not to allow the patient to see anyone except the doctor, the nurse, and the hospital attendants. Sometimes it is wise to prohibit correspondence. In less severe cases it is wise to allow the husband, or if the patient be an unmarried woman, some one of the family, to make short visits daily, or every few days, but all discussion should be tabu. Isolation should not ordinarily be kept up for a longer period than a month or six weeks. After that infrequent visits should be allowed and the patient should be encouraged to write letters and be allowed to receive mail. One would think that to put a patient to bed and to isolate her from everyone she knows would lead to great mental depression and do her injury. If this were all the treatment it would do much harm, and no good, and would have very evil results, but the time is so occupied by treatment during a rest cure that there is little time for the patient to get depressed.

The next important thing after isolation is bed rest. At first the patient should be strictly confined to bed, except that while the bed is being made in the morning she may recline on a couch. The time that she should be closely kept in bed will vary from four to six weeks. Then she should be allowed up fifteen minutes twice daily, then thirty minutes and finally she should be up three hours morning and afternoon.

Assuming that the patient has been taken to a rest house or hospital and isolated and put to bed, we come next to the daily routine of treatment. The day begins at about eight o'clock with a glass of cocoa immediately followed by a sponge bath in bed. There are a few women who cannot digest cocoa or whose dislike for it is intense. It may then be replaced by warm milk, followed by coffee, but some hot drink in the morning is very useful. Every two hours thereafter till eight or ten o'clock at night she should be given eight ounces of milk with one-half ounce of lime water to each glass. At first, and for several weeks, she should have no other food. Many women think they can not digest milk. In a few this is true but the great majority can, and most of the remainder can get along very well if lime water is added, or if they learn to take the milk slowly, or if it be partly predigested. We know so little about physiological chemistry that it can not be proven, but I am inclined to believe that milk acts in a chemical way, apart from its mere value as a food in aiding neurasthenics. In mild cases there is no need for an absolute milk diet but milk should always be a large part of the dietary.

The next most important thing after isolation and milk diet is massage. The time of the day that it is given is not of very great importance, save that often when given at night it aids in producing sleep, but it should be given at the same time daily. There are a few women who are made wakeful by massage. In such cases it should, of course, be given in the day. Very rarely one finds a patient who is made so nervous by it that it can not be given. It is then necessary to substitute mechanical massage by means of a vibrator. This is far inferior to manual massage but sometimes it must be used. At first massage should be very light, a mere stroking motion, over the whole body lasting twenty minutes. But the time should be rapidly lengthened, up to forty-five minutes or an hour, and after ten days the patient should be getting quite vigorous treatment. It should be given by a woman who has been properly taught. Amateur massage is useless. The patient should be silent during treatment and she and the masseuse should not occupy the time by gossiping. The masseuse herself should be a healthy, sane woman and should not have cold, wet, blue hands. The personality of the masseuse plays a large part in success or failure. She should not have opinions, at least ought not to express them, and should not try to amuse and interest the patient by relating tales about the desperately ill people she has cured. The patient is prone to remember the horror and forget the cure.

After massage comes electricity. The two should be given several hours apart, one say in mid forenoon, the other in the mid afternoon. The only apparatus required is a nurse's faradic battery. The entire body except the chest and face should be gone over with the slowly interrupted current, the poles being placed near together over the bodies of the muscles and each muscle made to contract three or four times. There is no need to put the poles on bony places; bones will not contract and are sensitive to pain. After all the muscles have been gone over, which takes about forty-five minutes, a medium-sized electrode should be put at the back of the neck and a large flat one on the soles of both feet, both well wetted with warm water, and the rapidly interrupted current passed for about fifteen minutes. Massage and electricity do good in several ways. Both give the muscles work without any nervous exertion on the patient's part. Both, and especially massage, mechanically squeeze out poisons from the muscles and both probably aid in increasing the flow of the current in the lymph channels. Both also have a purely psychical effect and finally they occupy time.

Medicine plays only a small part in the rest cure. It is necessary to aid the bowels because the inactive life in bed and the milk diet tend to cause constipation. Abdominal massage helps to overcome this but usually some medicine must be given. As a rule the fluid extract of cascara sagrada acts well. It should be given in one dose daily, at night. When cascara fails, senna or the alloin, belladonna, and strychnia pill often act well. It is not wise to use salines daily. It is very important not only that there be a daily stool but that it should be of good size. The nurse should always see the stool because often patients will report that their bowels are normal when as a matter of fact they have had a little, hard, and entirely insufficient stool. Indeed they may have a diarrhœa of constipation—a little watery stuff coming away each day but leaving behind hard lumps. It is wise, no matter how well the bowels may seem to be behaving, to give a high enema once each week. If cascara, senna, or the pill do not give good results it is better to give a daily enema than to use salts continuously.

The neurasthenic usually sleeps poorly and the question always arises whether to use drugs or not. Often massage at night or a hot bath or a pack will overcome the difficulty. Sometimes as simple a thing as a glass of hot milk suffices. When these fail, recourse must be had to drugs. Morphia must not be used, nor alcohol. Veronal in five-grain doses given at eight o'clock in the evening often acts well. If it fail, each of the hypnotics must be tried in turn till the one that acts is found. But beware of causing drug habits. All neurasthenics tend to depend on drugs and then we must cure them of the dependence. No hypnotic should be given unless the insomnia is so great as to show by emaciation and weakness that sleep must be had. The mere statement of the patient that she is sleepless means nothing. There is nothing in the treatment of neurasthenia that requires more care than the use of hypnotics and sedatives. The patients crave and long for something to give them sleep and to stop the depression and restlessness. If they are permitted to drug themselves, almost inevitably they will get some drug habit. Much harm comes from the ease with which veronal, trional and similar drugs can be bought. Giving sedatives to neurasthenics is a much more serious matter than many people realize. The temptation to use them is very great as the temporary relief to the patient is marked, but really they do harm except when absolutely necessary. It is very common for a neurasthenic while still up and around to dose herself with bromid and veronal or some other like drug and soon she becomes entirely dependent upon them. I have more than once seen women who kept themselves in a permanent condition of semi-stupor by drugs. Rather rarely women neurasthenics are secret drinkers. They often use bitters, or quack medicines, or cologne. One case took large quantities of tincture of valerian and finally was seized with an alcoholic multiple neuritis. The morphia habit is not common among neurasthenics. Every patient, however, who seems to be a neurasthenic should be examined to discover if she is a drug habitué.

It is best to give no drugs for the restlessness and irritability present in the day time. It may be necessary to use bromid, but it should not be given if it can be avoided. It will stop restlessness but it is very depressing and hence has an evil effect. Tonics are valueless, except a little for the stomach's sake, and the old-fashioned nux vomica and gentian is the best. The patient should never know what medicines she is getting.

If the rest cure is going to do good there should be improvement in four to six weeks. By that time the pallor should have left the skin, sleep should be better, there should be desire for food, the bowels should be in good condition, the nervous restlessness should be much less, there should be times when the depression lifts, and the patient should begin to resent being in bed and to want to do things. She should begin to show interest in the outside world. Now the patient should be allowed to see her people, to read a little, to write letters and get up. At first she should be up fifteen minutes twice daily, then thirty minutes, then an hour until finally she is in bed in the daytime only during massage and for an hour later. Electricity may be stopped and the diet gradually enlarged until she is on ordinary food. Now comes the time for a work cure precisely the same as described under psychasthenia.

I wish to enter a word of protest against the Freudian treatment of neurasthenia and allied affections by psychanalysis. The theory is that many of these disorders are due to unconscious, repressed wishes, often, if not always, sexual in nature. The alleged origin of much trouble dates back to thoughts and habits in early childhood. Now by free association, by word association and by symbolic interpretation of the patient's dreams the unconscious wish is, it is alleged, uncovered and when it is pointed out to the patient that she is really suffering from a conflict between complexes in her unconscious mind she is supposed to recover. A great deal of the treatment consists in talking sex matters with the patient. This does much harm and no good. I have seen much evil result and little good from Freudian psychanalysis. A frank, wise talk, once for all, often helps the really sexually ill neurasthenic.

Every case of neurasthenia does not need a complete rest cure. Many patients if seen in the early stage of the illness can be cured by a combination of rest and exercise and play. Such patients should be taken from home, made to rest one hour in the afternoon, go to bed early in the evening, and stay in bed till after breakfast, say till nine o'clock. If they do not know how to play they should be taught some out-door game. The kind of game depends upon the physical strength of the patient. It may be lawn tennis, croquet, or golf, or the gentle sport of fishing, depending upon the strength and opportunities of the patient. They should read little and think not at all. They should have a shower bath daily followed by a rub down. No drugs should be given. Much harm comes from putting patients to bed who ought to be kept out of bed and from making people work who ought to rest. Skill is needed to determine in each case what is needed. The failures following rest cure have frequently been caused by error in judgment as to this matter of rest versus work.

Psychasthenia.—It must be understood that the word psychasthenia is used here in a different sense from the common one. It is ordinarily used now as synonymous with neurasthenia. The tendency is to regard neurasthenia and several other disorders as really a psychasthenia and to replace a rest cure by a work cure. One reason for this change in opinion is the fact that after the rest cure became popular it was often used in unsuitable cases, in cases that needed exercise and diversion instead of physical rest and mental inaction. The result was evil and a method that was intended for one condition was blamed unjustly because it did not produce good results in another requiring an entirely opposite mode of treatment.

There is a psychasthenia apart from neurasthenia, a condition of pure mental tire, from overwork and excessive strain and resulting in inability to do mental work. It is a less serious affection, and much more dependent upon external causes, than neurasthenia.

The hereditary element as a causative factor is much smaller. There are few if any truly physical symptoms. It is most common among intellectual workers and persons under emotional strain. It is more frequent in men than in women. There are many cases of mixed neurasthenia and psychasthenia, but both affections occur separately. Psychasthenia, as here defined, much more nearly resembles physiological mental tire than neurasthenia resembles normal nervous tire.

Symptoms.—The onset is always slow though after an unnoticed prodromal period there may be a sudden outburst of symptoms, a sudden breakdown. The patient finds that he is not able to do as much mental work as before. He can not keep his attention fixed. Unlike the neurasthenic he, at the beginning, wakes in the morning not depressed but refreshed and feeling able to do the day's mental work, but soon he finds he is exhausted. As he grows worse, however, he has the same experience as the neurasthenic, namely, he wakes tired, mentally, weary and unrefreshed, yet in the evening, notwithstanding the hardship of the day, he is mentally alert. One of the early symptoms is difficulty in quickly changing the subject of thought. For example, if he is intensely thinking on a subject and is interrupted by some outside thing requiring him to think of something else, there is a perceptible interval of time before he can get his mind on the second thing and another interval, and perceptible effort, before he can return to the first subject. There may even be a moment of mental confusion or even vacuity before he can take up the second line of thought, and again, when he returns to the first. There is always more or less loss of power of recollection of recent events. The patient will forget whether he has written a letter, signed a check, paid a bill and such things. There may be no true memory loss, the imprint having been made in the brain, but the patient being unable to recall it to consciousness. On the other hand sometimes, from disability to fix attention, things may not fasten themselves in the brain and hence a true memory loss results and not merely lack of power of recollection. The patient can not compel his mind to do his bidding; he can not hold it to the work he wants done. There are few purely physical symptoms in psychasthenia. The most frequent one is nape-ache, or, in the language of the patient, pain at the base of the brain. There may be loss of appetite, but there are never the symptoms of active indigestion common in neurasthenia. Physical tire is not prominent, but there is psychical inertness or rather inertia of the will, making it very hard for the patient to take exercise or do physical work, but if forced to it he can do it well and without any sign of cardiac weakness or dyspnea. In neurasthenia there is true physical weakness as shown by rapid heart, short rapid breathing, a leaking skin. In profound psychasthenia there is always emotional depression. It is caused usually and in great degree by fear. Nothing frightens a man more than to realize that he can not think and plan and attend to his work. He immediately begins to fear insanity and thus a vicious circle is established. Psychasthenia is a disease of mature life. It occurs either in men who are carrying a heavy load of responsibility or a load relatively heavy for their power. It is more common in men than in women but as women increasingly invade man's sphere it is probable its frequency in them will increase.

Diagnosis.—The most frequent error made is to regard a case of beginning paresis as one of psychasthenia. Paresis is usually insidious in onset and as a rule for weeks or months relatives and friends think the patient is only run down from overwork and worry, and is suffering from mental fatigue. They have not the slightest suspicion that anything is seriously wrong until after the prodromal stage is passed and irreparable injury has been done to the cerebral cortex. Early diagnosis is important because early treatment might stop the progress of the disease even if it did not cure the illness. The points of similarity between psychasthenia and paresis are the following: rapid mental tire, inability for continuous thought, inability to fix attention, and loss of power of judgment. In paresis there is no realization of ill health except that at the beginning the patient may dimly realize that there is something physically wrong. Very soon, however, this feeling, if it has existed, passes away and the man indignantly denies that there is anything the matter. Very rarely the paretic fears approaching insanity before manifest symptoms have appeared; the psychasthenic fears all the time. The psychasthenic knows only too well that he is ill. In paresis there is always, sooner or later, and as a rule before any mental symptoms occur, symptoms pointing to organic disease of the cerebro-spinal-axis. One of the commonest is a transient oculomotor palsy. Sometimes the illness may begin, though this is rare, with an epileptic or apoplectic attack. One or both frequently occur sometime during the course of the disease. In paresis there are always, but not necessarily at the beginning, organic disturbances of the reflexes, of articulation, and of gait. None of these occur in psychasthenia. There may be an emotional (hysteric) disorder of gait, but it is so manifestly dependent on mental conditions that no one could confuse it with an organic palsy or ataxia. In psychasthenia, as in many functional nervous disorders, there may be a tremor of the hands but it is emotional and entirely unlike the intention tremor of paresis. Syphilis practically always precedes paresis and as a rule there is a history of high living. Error in diagnosis can only be made in the beginning. But such error can always be avoided if it is remembered that, given a man of mature years, the existence of oculomotor palsy, of scanning or slurring speech, of epileptiform or apoplectiform attacks disproves absolutely mere psychasthenia and indicates paresis. The mental tire of the psychasthenic can not be mistaken for the dementia of the paretic, if it be remembered that the former thinks normally, though thinking makes him tired, while the dement always thinks abnormally. Of course the presence of delusions of any kind disproves psychasthenia and proves insanity.

Treatment.—A neurasthenic needs rest, a psychasthenic exercise. A neurasthenic should have no work, a psychasthenic needs change of occupation. Travel hurts a neurasthenic; it may aid a psychasthenic. Assuming that a patient is so situated that he can do just what he wants, the best thing is, if the case be a severe one, to leave business entirely, leave cities and the haunts of men and go on a camping expedition. A trip to Europe may be good, but travel should be slow and leisurely. Rushing around the world always does harm. Milder cases do not need such rigorous treatment. If the patient will give up, say, two hours daily to active exercise and will give up alcohol, this may be all that is needed. Alcohol is bad, even in great moderation, for either a neurasthenic or a psychasthenic. It often gives temporary relief and enables a man to cross a bridge, but the reaction is inevitable and severe. Tobacco is much less injurious. Theoretically it should be discontinued, but indulged in moderately (unless a tobacco heart be present) it may be permitted. Many a psychasthenic of sedentary habits and leading a too intellectual life has been cured by such a simple thing as going on a farm and cutting down trees till he is so tired he can not stand.

There are cases of mixed neurasthenia and psychasthenia in which a combined rest and work cure is needed. The mixed condition is very common in women. After the physical weakness has been relieved by a rest cure there comes a time when the patient must be put to work. In such people there is always a total lack of interest, a loss of all ambition, and it is just as important to stimulate interest as it is to have a certain amount of physical work done. The institution of occupation rooms in hospitals for nervous diseases has done much to help these patients. Women can be taught to do basket weaving, modelling in clay, all kinds of fancy sewing, decorating. These things can all be done in an institution. For men the best thing is out-door work, gardening, boat building, or any kind of carpentering work or even chopping wood. No man or woman is so old but what there can be aroused in him interest in some kind of handicraft or some kind of out-door play. Among games golf is the best for these people. It is the one game young and old can play. It is not too strenuous for even the septuagenarian; it is not too tame for the youth. But no good comes from giving an invalid a golf ball and a set of clubs and sending him out on the links. He should be taken in hand by a teacher and taught and made to do it as earnestly as he would a very serious business matter. The important thing is to arouse the patient's interest in the matter because the philosophy of it is to strengthen the mind by exercising it in new ways.

Hysteria.—Certainly in America, probably everywhere, hysteria is a rare disease. Hysterical symptoms occur in, often are a part of, the symptomatology of many organic diseases, especially those of the cerebro-spinal-axis, but hysteria occurring alone and not as a complication of something else is infrequent. Its apparent great frequency is largely to be accounted for by the fact that for some years it was more written about and studied than any other nervous disease. An unconscious mental result of this upon physicians in general has been to make them assume it is a very common disorder. Another reason for its apparent frequency is carelessness in diagnosis. Too frequently the diagnosis is based on the mere presence of unusual or bizarre symptoms without any endeavor being made to discover if there are not present organic causes. The word hysteria is used too loosely. It should be strictly limited to mean only palsies, convulsions, disturbances of general sensibility, special sense disorders, and mental symptoms of clearly defined types, and not used as a label for all sorts of queer and unusual nervous conditions.

Because a person is of the hysteric temperament she or he is not thereby protected against organic disease. A very common error is to be so struck by the existence of some hysteric symptom as to overlook signs of organic disease and to regard the illness as being wholly hysteric. Hysteric symptoms in organic disease of the nervous system are very frequent in women and not infrequent in men. Almost every case of tumor of the brain in women I have seen, at the beginning has been diagnosed hysteria, or at least grave question has arisen as to whether a tumor really existed. Thus some years ago a woman complained of diffuse pain in the right arm. She showed no objective signs of organic disease and was very hysterical. For months she was treated as a hysteric, but without result. At the end of a year she was brought to see me. The picture had changed a little. She now showed a little wasting in the muscles of her right thumb, and indeed of the whole hand, and her right arm was weaker than the left. She could move the arm, hand and fingers in all directions but all the movements were paretic. The pain continued. It was not confined to the distribution of any one nerve trunk or combination of nerves but affected the arm as a whole. Both nerve trunks and muscles were very painful on pressure. I made a diagnosis of neuritis involving the brachial plexus but I could not find out the cause. Nothing that I did for her helped her in any way. She grew more and more nervous, at times was quite hysterical in the popular sense, and the wasting and palsy of the arm steadily increased, the pain continuing. After several months she rapidly, indeed acutely, developed symptoms of disease of the bulb and died. At autopsy we found a long, flat subdural tumor on the right side in the cervical region of the spinal canal, extending up to the bulb. Undoubtedly the tumor had first caught a number of the sensory roots and squeezed them enough to cause pain. Later the motor roots were also involved and brought about the palsy and wasting. Another case was that of a woman who in addition to many hysteric symptoms had convulsions affecting the whole left side without loss of consciousness. There was a difference of opinion for months as to whether the Jacksonian fits were also hysterical. The question was settled by the appearance of choked disc. She had a tumor of the brain. The correct diagnosis, or at least a diagnosis of organic focal disease, ought to have been made from the first because true Jacksonian fits are not a part of hysteria. Too much stress was laid upon the absence of choked disc. Hysteria is almost as frequent in men as in women. There are distinct racial predispositions. Thus the Jews, both men and women, are especially prone to it or rather are prone to it as a complication of organic disease. The French have long been regarded as preeminently victims, but it is probable that its seeming frequency among them is really only seeming and due to the amount of time given to its study in France. It is increasing in frequency in America probably because of the character of the immigration at the present time and also on account of the changing attitude toward life that is taking place. In diagnosing hysteria it must never be forgotten that the disease never begins in middle life. It is a disease of the young, just as much as neurasthenia, i. e., it always begins in adolescence or early maturity. Of course attacks may begin at any age, but if a correct history is obtained previous attacks will always be revealed. It sometimes occurs in childhood. The commonest form in children is the convulsion and the next most frequent blindness, either unilateral or bilateral. Hysterical palsies may occur in children but they are not as frequent as convulsions, and mutism is occasionally seen. The common practice of diagnosing an illness in which there is only one symptom, say palsy of the leg or arm or pain in one part, as hysteria leads to much error. Monosymptomatic hysteria occurs only in children. They may have only convulsions, or blindness, or an hysteric joint, or a mono- or paraplegia, or mutism. When hysteria occurs in adults there is always a more or less complete picture, or a history of the previous occurrence of other symptoms. A healthy adult, of previous good health, is never seized by hysteria presenting only one symptom, but an adult who has had previous attacks may show only one symptom.

There has been much discussion as to the essential nature of hysteria. For years it was regarded as a palsy of the will. For years everyone quoted the maxim "it looks like I will not, it really is, I can not will." Today there are two much discussed theories: one that it is due to suggestion, the other that it is really sexual in origin. Much is to be said in favor of suggestion but I am not altogether convinced that suggestion is the sole and only exciting cause. Everyone admits that the real cause, the predisposing cause, is inherent. Those who believe in suggestion as the exciting cause of the symptoms explain the strange contradiction, that an hysteric who has a one-sided anæsthesia may still be able to use the anæsthetic hand and recognize objects put in it, by saying that there is no real anæsthesia and that the apparent anæsthesia is produced by the examination. But this does not explain why hysteric anæsthesia is most often left sided. Again, unless it were done purposely, it would be difficult for geometric anæsthesia to be due to suggestion. If the convulsions were caused by suggestion they would vary in nature. It would not come to the mind of every woman to convulse in the same way and yet the picture of an hysteric convulsion is definite. Physicians who act as experts for railways have been quick to seize this explanation of hysteria. Such a cause would relieve railroads and other corporations from responsibility, it being, I believe, a rule of law that damages can not be gotten for mental anguish not caused by physical disease, and of course a symptom due to suggestion would be purely mental. The truth of the matter is, it seems, that some hysterics are very suggestible and will pick up symptoms if given an opportunity, but that others are not at all suggestible. After all, suggestion does not explain very much because then the question would be what makes hysterics suggestible.

The theory of the sexual origin of hysteria has been carried to an extreme. That there is a large sexual element in it is undoubtedly true. Many hysterics, male and female, have been excessive in intercourse, some have unnatural habits, some have a craving they do not recognize the nature of, and in some the sexual feelings are normal in nature but excessive in amount. But hysteria is not, as is claimed by some writers, simply the expression of an unconscious, repressed sexual wish. Nor can it be cured by discovering shameful and painful events in the past history of the patient, either from her history, her dreams, or the blocking of word association and then showing her what the matter is. Barring the cases in which there is unrecognized sexual hunger, the sexual element is not unconscious or subconscious but very conscious. So far as symptoms go there may be no sexual manifestation of any kind. It must be remembered, however, that many patients suffer greatly from sexual excitement and for very natural reasons do not speak of it.

The real cause of hysteria is entirely unknown. It is surely something which affects the whole organism and influences the entire personality of the patient. Its origin is congenital. Hysterics are born not made. It is not caused by external stresses and strains. They only give it opportunity to manifest itself. When we discover what is the cause of personality, what is its physical basis, then we shall know what hysteria is because it is a disease of personality. As yet we are far from having made this discovery.

Differential Diagnosis.—The great fact in hysteria is the presence of physical symptoms referable in great part to the central nervous system but which may involve almost any organ, in the absence of organic disease of the cerebro-spinal-axis or other viscera, with or without definite signs of mental disorder. In hysteria the impossible happens. Symptoms that can not occur do occur and are not merely simulated. Hysteria is bizarre, but that very thing sometimes leads us to overlook an accompanying organic disease. Because hysterical symptoms are present one must not hastily conclude organic disease is absent, but prove its absence. Though the symptoms may be bizarre they are never thrown together helter-skelter; there is always a method in their arrangement. Certain symptoms are always grouped together.

One of the most difficult problems is to differentiate hysteria from the insanity of adolescence (dementia præcox). Indeed one is inclined to believe, sometimes, that each is but a different presentation of one disease, and that the different pictures, when they are different, is simply due to differences in the personality of the victim. Whether this is true or not it is customary to separate the two conditions and in their extremes they are not difficult to separate. The trouble comes when the hysteria manifests itself mainly by symptoms that are purely mental. Thus in both there may be hysterical convulsions, mutism, and transient palsy. In both there may be trance states. It is safest if delusions be present, even transitorily, to regard the case as one of insanity, though even then errors may occur, but when they last many days insanity is surely proven. The importance is in the matter of prognosis because true adolescent insanity always ends after, at the most, one or two intermissions in permanent dementia, while hysteria may recur many times throughout a long life and yet the patient be entirely well, or at least regain her normal, after every attack.

The differentiation of hysteria from organic nervous disease may be easy or difficult. Certain general rules help. Hysteria is always multiple in its symptomatology. It never begins in middle life or later. There are a few symptoms that prove organic disease and, if organic disease be present, the existence of hysterical symptoms is of no importance, because it is the organic trouble which must be treated, the hysteria is merely symptomatic. True ankle clonus never occurs in hysteria. There may be an aborted clonus; the foot may give a few irregular jerks when it is passively, strongly flexed dorsally, but the movements are not equal in extent, do not follow at equal intervals, and in a very short time cease. A true Babinski jerk is never seen. On stroking the sole the whole foot may be forcibly withdrawn and in doing so it may flex dorsally and the toes extend, but there is never slow extension and fanning of the toes. The most characteristic knee jerk, but by no means pathognomonic because it occurs in many conditions of nervous irritability, is a sudden large extension of the leg with a start of the whole body. The knee jerk is never absent.

There are certain sensory symptoms which are pathognomonic, viz., gauntlet anæsthesia and geometrical anæsthesia. In the former, one extremity is anæsthetic up to a certain level without regard to nerve distribution or cerebral sensory centers. In the latter there are scattered over the body areas, sometimes accurately geometric in shape, that are anæsthetic. Hysteric hemianæsthesia stops with a clear cut boundary exactly at the middle line. Hysteric amblyopia is fairly characteristic. Reversal and contraction of the fields of vision, which formerly was thought to be pathognomonic, is almost as frequent in tumor of the brain, independent of location, as in hysteria. Tubular vision, however, is always hysteric. A field which decreases during examination is significant of nothing more than tire (neurasthenia). Monocular diplopia, not caused by a dislocated lens, or some other obvious physical defect, is always hysteric. Sometimes in hysteric hemianæsthesia there is blindness in the eye, deafness in the ear, and loss of smell and taste on the anæsthetic side. This is a combination of symptoms which can not be caused by any organic disease anywhere in the brain. True aphasia never occurs in hysteria but aphonia is common and mutism is not rare. The aphasic can be differentiated from the mute by the fact that the former always can make sounds of some kind and manifestly tries to speak while the hysteric mute makes no sound and does not try. Also the accompanying symptoms in the two conditions are different.

The hysterical convulsion is characteristic. Consciousness is never lost. The patient does not fall, but sinks to the ground. The tongue is never bitten though the lips may be. There is never relaxation of the sphincters though after the attack is over the patient may, and frequently does, pass a large amount of water. The convulsive movements are always histrionic, and may be mystically-religious or sexual. The patient may act out a whole pantomime in her convulsion, showing love, hate, and adoration, but such cases are, in this country, very rare. The movements of true epilepsy are wild, incoordinate and manifestly unwilled. Consciousness is lost, the spasm is first tonic then clonic, a cry precedes, the sphincters may relax, breathing is stertorous, the face blue-red, or almost black, blood comes from the bitten tongue mixed with the frothy saliva. The epileptic fit is short, the hysteric prolonged. Deep sleep follows the epileptic fit, but not the hysteric. Petit mal is never hysteric. No matter what hysterical symptoms a patient may have, if she has attacks lasting only a moment, during which she is dazed, stops talking and drops whatever she may have in her hand, the case is one of true epilepsy. It is important to remember this because many epileptics are also hysterical and as people pay no attention to the little attacks, which they regard as unimportant, a true epileptic may for years be regarded as only a hysteric. The word hystero-epilepsy to indicate major hysteria should not be used because hysteria and epilepsy are distinct disorders and when they are combined it is mere coincidence.

There is no way to differentiate the trance of hysteria from similar states in dementia præcox save by the previous history and the future conduct of the patient. Fortunately the treatment is the same for both.

Hysteria may present a picture simulating multiple sclerosis. The chief differentiating points are that in hysteria the onset is sudden, the nystagmus is not like that of organic disease, the symptoms are exaggerated, that is, too marked, and the cure is sudden. The onset in sclerosis is always slow or at least not sudden. There are never convulsions in multiple sclerosis, nor true scanning speech in hysteria.

The differential diagnosis from malingering is important because there is probably no disease more frequently simulated than hysteria. It is the only disease ever simulated for no other motive than to gain sympathy, the pretender having no other object than sympathy itself and knowing that no material advantage will be gained. To crave sympathy, to desire to have a fuss made over her, is part of the temperament of many, but not all hysterics, and this leads to much simulation. This does not mean that hysteria is fraud nor that all hysterics are malingerers. On the contrary it is a real disease and many hysterics make every effort to get well, but there are people, hysterics, whose desire for sympathy is in itself a symptom of disease.

To come to true malingering. There are many reasons for its frequency. The pretender always presents symptoms so striking as to attract attention and to make the casual observer think he must be dangerously ill. Pretended symptoms are always disabling. Thus a paralyzed soldier can not be expected to go on duty. A prisoner who is subject to fits can not, today at any rate, and with the present attitude of the public as to our duty toward the unfortunate classes, be left in a cell by himself. He must have a cellmate or be kept in the hospital ward. The petty criminal may escape punishment altogether by a well feigned hysteric attack in the dock. He is bound to get the sympathy of well-meaning, sympathetic and ill-trained or untrained probation officers. In recent years, with the increase of machinery of all kinds, and the increasing responsibility put upon employers for the welfare of their employees, there has been both an increase in the number of accidents and an increase in the number of cases of real traumatic hysteria and of fraud. The same is true concerning railroad accidents. The increasing responsibility of the companies to their patrons increases the number of fraudulent charges made against them. One of the unforeseen results of state insurance also has been the increase of fraud. "The state is rich, I am poor, why should it not support me?" This is the argument.

Every symptom of hysteria may be more or less successfully simulated. Probably the fit is the easiest and the most frequently attempted. Then follow palsy, especially paraplegia, and last, disorders of the special senses, especially blindness and deafness. The trance state and living without food are occasionally simulated, not, as a rule, to get money for alleged injuries, but to receive money by exhibition or merely to create a sensation. Multiple personality, which when real is an hysteric manifestation, is occasionally simulated because there are worldly reasons why it should happen. Sometimes the game is played on physicians who are interested in the subject either for the money the pretender hopes to make out of them or for a more elusive reward, namely, to be immortalized in a learned treatise. Sometimes it is of very practical use to the pretender to disappear from view and live somewhere else as somebody else. The skill rather of a detective than a man of science is needed to unmask a false case of double personality.

It may be easy or difficult to diagnose malingering. Pretended blindness and deafness can always be discovered by special tests. Palsies never present the proper accompanying symptoms. The fits are prone to be crude. A practiced eye can recognize them. As to alternating personality there is no objective sign by which its reality or unreality can be proven. If the patient be carefully watched he usually will show, if he is a fraud, by word or act that he remembers something that he ought to have no recollection of. A very simple test to prove fraud in pretended trance is to watch the bladder and bowels. The fraud will drink no water and pass the usual quantity. He will eat no food and have a daily stool. The length of time a person may go without food without dying varies with the individual. If given water the time is lengthened.

Treatment.—The rest cure is the best treatment for the majority of the cases of hysteria. Pretty much any kind of treatment may cure an attack, and this explains why quackery flourishes, but to cure the disease is very difficult and often impossible. Convulsions, palsies, anæsthesias, and joint troubles often cease suddenly and under the influence of any strong mental influence. All kinds of psychotherapy have been claimed to cure hysteria and undoubtedly they often cause the symptoms present at the time to disappear, but they rarely change the nature of the person. Hypnotism has had large vogue as a cure, but on the whole it has probably done more harm than good. Its apparent therapeutic effect may be very dramatic but its influence, especially if long continued and repeated, is evil and not good. Electrical treatment of any kind may make many hysterical symptoms pass away. It is not the electricity but the mental influence which causes the cure. Unfortunately these methods do not eradicate the disease but only make the patient better temporarily. The rest cure sometimes, on the contrary, has a real curative influence. There is in it a psychic element but it is not all psychic; part of it, and a large part, is material.

More important than cure in hysteria is prevention. This is a matter of education, not in book learning but in how to live. The boy or girl of an hysteric temperament can, if brought up under proper conditions and trained to look at life from a normal point of view, be enabled to escape the destiny which his ancestry has prepared for him. Especially is it necessary to train such youths in self control and emotional balance.

Borderland Mental and Nervous Disorders

There are a number of people who are neither sane nor insane and another group who, though not suffering from any distinct and definite nervous disease, are yet neurotic and nervously unsound. The two groups make up the borderland population of the country of ill health. It is often impossible to label the individual sufferers with names for their ailments. Some are almost true neurasthenics, others just escape coming within the boundaries of true insanity. In some cases the symptoms are almost entirely nervous, in others wholly mental, and in still others, the largest number, there is a mixture of nervous and mental elements. All are alike in that heredity is the great causative factor, environment playing only a subordinate part, and in that, as a rule, the condition shows itself early in adolescence and increases in intensity as time elapses.

The symptomatology of the neurotic would take many pages accurately to describe. They are bastard sisters and brothers of the neurasthenic and hysteric. They show symptoms of both conditions and yet are not classical examples of either. They probably are people who would have been one or the other had their personalities been different. Mere neuroticism, neurasthenia, and hysteria are all three diseases of personality and which condition shows itself in any given person depends upon the individuality of that person. The central symptom of neuroticism is excessive irritability, excessive sensitiveness to all stimuli combined with extreme egotism. This purely mental quality, egotism, is present in most patients even when their complaints may be wholly of physical pains and disturbances. There is no clear cut line between the neurotic and the psychotic. They run into each other. Many of the neurotic resemble hypochondriacs in that they complain of bodily ailments that have no real physical existence but they do not have definite delusions concerning their organs.

The usual history is that a girl, for females are more prone to neuroticism than males, who has always been guarded and secluded, often because her people have regarded her as delicate and have attempted to strengthen her by keeping her protected from stress and strain, rather than making her strong by subjecting her to proper discipline, begins to complain of many aches and pains. Pretty soon she takes advantage of her condition, it may be unconsciously or subconsciously, to impose on others. Noise distracts her, so there must be no noise. Certain foods she can not eat, even bear the sight or smell of: they must be banished from the table. Other food she is fond of: it must be bought even though the family pocketbook can ill afford the expense. She can not bear the heat of summer, or the winter's cold: she must be sent away at whatever cost. As time goes on her symptoms increase. Many doctors see her; many kinds of treatment are used but the illness continues. She grows worse and worse and symptoms affecting all or almost all organs of the body appear and yet examination reveals nothing. She needs more and more care and attention and more and more does her family become her slaves. Now she has attacks of violent headache, or sick stomach or general weakness and periods of faultfinding and even anger. She may have, often does have, unpleasant sexual symptoms and may have developed the habit of masturbation. There may be only unconscious, normal sexual hunger. Her life is miserable. She can not work. She can not play. To read causes tire and headache. She loses all interest in life and her world narrows more and more until life is scarce worth the living. Now all this looks like mere selfishness and one is prone to deny the existence of disease, but really the selfishness is truly pathological and is a sign of deep-seated disease.

The purely mental borderland states are more definite. They can after a fashion be classified. We may divided them into: 1. The eccentric, 2. the paranoid, 3. the obsessed.

The eccentric are the most numerous, the least interesting, and were it not for the fact that they are more prone than normal people to have degenerate offspring, are indeed the greatest source of degeneracy, because they live in the outside world, marry and beget children, while large numbers of the insane are protected against themselves in institutions and others are sterile, the least important dwellers in the borderland. I am speaking only of the congenitally eccentric, those people who very early in life, even in early adolescence, show signs of looking at life from a point of view different from that common to the time and place of their abode and not consonant with their education. Acquired eccentricity coming on in old age, a manifestation of the normal and mental breakdown of senility, is only a part of senile dementia and does not signify any previous weakness in the sufferer nor increase the danger of his children being abnormal in any way. Congenital eccentricity may be so slight as to be trifling. Many of us have little eccentricities which give pleasure to ourselves and result in no harm to others. This leads most people not to regard eccentricity from the point of view of disease and not to take it seriously, but looked at from the psychological point of view, which is the correct one, marked eccentricity is pathological, is really a mental disease. The central psychological cause of congenital eccentricity is pathological, mental independence, or in other words intense belief in one's own infallibility coupled with a carelessness as to the feelings and opinions of others. It is a form of intense egotism. It may be associated with extreme shyness though this is often hidden by an affectation of brusqueness. Its victims rarely have much social instinct and usually care little for the society of any people save those whom they can dominate. Slight eccentricity we may dismiss but the serious cases are well worth study. They have no realization of the relative value of things and put great emphasis on matters that the world regards as of no importance. In some their slavery to what they think is logic and their belief in the great importance of consistency leads to much trouble. There is no need to describe eccentricity because it is too well known. Its importance is its influence on future generations and the possibility of its prevention by education and training.

Paranoids.—The paranoid is an aborted paranoiac. He is dangerous to the community. There is no clear cut dividing line between the sane and the paranoid, nor between the paranoid and the true paranoiac. The great symptoms of the paranoid state are egotism, extreme vanity or conceit, and a feeling of persecution. The paranoid never develops a clear cut delusion of persecution but he always feels that the world does not treat him right, does not give him his just dues, and does not shower upon him the rewards he deserves. He has a moral code and follows it, but it is one sided. It contains many laws about other people's duty toward him but contains nothing about his duty to other people. He is always intelligent and sometimes superficially brilliant, but his mental processes are always twisted. He is mentally perverted and looks at everything through distorting glasses. He is morally perverted as well. He is not industrious in productive work but very busy in useless or harmful things. Just as in true paranoia there are mystics, and politicians and men of science, i. e., people whose delusions concern these matters and who think they are the masters and leaders of the world prevented by a conspiracy from being given their proper place, so is it among the paranoids. They are dangerous to the community because they may do some criminal act, even murder. They are dangerous to the family because they destroy all happiness and make slaves of everyone dependent on them.

The obsessed are very numerous and often are unsuspected by anyone of being in any way abnormal. An obsession is an idea imperiously demanding notice, the victim being painfully conscious of its domination over his work and will. It differs from the *idee fixe* in that it is not a delusion, the sufferer not being deluded, not, e. g., having any idea of persecution. He knows the unreality of the idea but can not escape from it. Obsessions occur at all ages from childhood to old age. They are a sign, of course, of degeneration and in the young are sometimes the forerunners of serious mental trouble in later life. An obsession in the aged may be the first symptom of senile dementia. On the other hand, obsessions may be mere trifling annoyances. Thus all of us have been worried by the fear that we have not locked a door though we know we have, or that we have not properly answered a letter though we know the answer was promptly written. They vary in intensity from these trifling things to obsessions that drive to suicide, so great is the mental pain. They may be transitory or permanent. Many fears are really obsessions. Thus agoraphobia, claustrophobia, acrophobia, astraphobia, zoophobia and the whole list of fears are all obsessions. Coprolalia is in the same class. Some victims must argue with themselves about the most absurd subjects.

Treatment of borderland states is largely preventive. It is a question of trying to overcome the evils of heredity by wise environment, i. e., by putting the child into the best possible surroundings—physical, mental, and moral. All three are equally necessary. It does not matter how good the physical environment of the child may be if his mental surroundings are poor, and his moral surroundings weak. The prevalent custom of trying to make children strong by shielding them from stress and strain will fail, because it only increases their weakness and when stress comes they will break down. The common notion that book education is the cure for all ills is equally fallacious, because an intellectual training in the absence of moral training only increases ability to do evil. Good training then is the only way to help children or youths who else would develop into borderland states of mental disease.

As to the treatment of the disorders themselves. A combined rest and work cure helps the neurotic. For the obsessed little can be done unless the obsession be really only a neurasthenic symptom. Fortunately many cases are cured by time. As to the paranoid, the only thing to do is to certify them as insane as soon as they begin to show dangerous symptoms, i. e., symptoms making them dangerous to the community. A physician should fortify himself well with evidence before signing a certificate of insanity or he may find himself the victim of a lawsuit.

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